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STRUCTURE AND COMPOSITION OF SALIVARY CALCULI.*†‡

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INTRODUCTION.

Salivary calculi are composed of an inorganic crystalline body and an organic matrix. This report is concerned with the identification and amount of the inorganic crystalline substance and the histologic, histochemical and biochemical characteristics of the organic matrix.

METHODS.

A total of 58 calculi surgically removed from the salivary glands have been included in the present study. Analyses were conducted over a period of five years, and hence not all calculi have been subjected to all analyses. The small size of many calculi precluded complete analysis of each stone. In

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general, calculi weighing more than 150 mg. were analyzed separately and smaller calculi were pooled for analysis. The average dry weight for the 58 calculi was 270 mg. with a range of 27 to 5670 mg. The inorganic constituents were determined by combinations of chemical and spectroscopic analysis.[1,2] The organic matrix was recovered by diffusion chelation of the inorganic constituents in 5 per cent EDTA adjusted to pH 7.8. Whole matrices of 14 calculi were fixed in 10 per cent formalin, embedded, serially sectioned to a thickness of 8 mμ. and studied by the following stains and histochemical reactions: *1.* hematoxylin and eosin; *2.* periodic acid Schiff leucofuchsin reaction; *3.* aqueous toluidine blue, Sudan black,

TABLE I.

Approximate Inorganic and Matrix Composition of 34 Salivary Calculi (Expressed as Per Cent of Dry Weight).

Constituent	Average	Range
Calcium	31	26 - 36
P (As Phosphate)	48	41 - 54
Organic Matrix	18	8 - 38

Magnesium, carbonate, and ammonium were present in small quantities but were not quantitatively determined.

and *5.* Von Kossa method for sites of "calcium binding activity."[3,4]

The pooled matrices from 12 of the larger calculi were recovered by lyophilization, hydrolyzed and examined by paper chromatography for amino acid and carbohydrate constituents as previously reported.[5]

RESULTS.

Inorganic Composition. Table I is a summary of the inorganic and organic matrix composition of 34 calculi. Calcium in the form of calcium phosphate (probably hydroxylapatite) was the principal constituent of all calculi. Magnesium was present in the range of a trace to 2 per cent (wt./wt.) of all calculi. Trace amounts of ammonium ions were detectable in all calculi, but oxalate was not identified in measurable quantity.

The matrix content of 18 per cent for salivary calculi is considerably greater than the 3 per cent of the average urinary calculus. Urinary calculi have been found to contain as much as 66 per cent by weight of matrix, and such calculi are roentgenolucent by ordinary diagnostic techniques.[4] No roentgenolucent salivary calculi have been analyzed in the present series. No salivary calculus has been found to be free of an organic matrix.

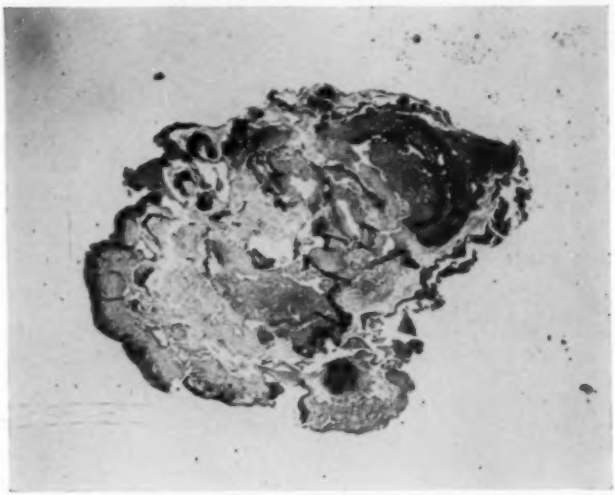

Fig. 1. Cross section of decrystallized submaxillary calculus. PAS stain. Magnification x 22.

Organic Matrix. As in urinary calculi the diffusion chelation technique for removal of the crystals has left the matrix as a perfect cast of the original calculus in every specimen (see Fig. 1).

Histologic and Histochemical Characteristics. The organic matrix is present from center to surface of all calculi. The matrix stains poorly with hematoxylin.

The periodate Schiff leucofuchsin reaction is brilliantly positive for the entire matrix (see Fig. 2). Treatment of the

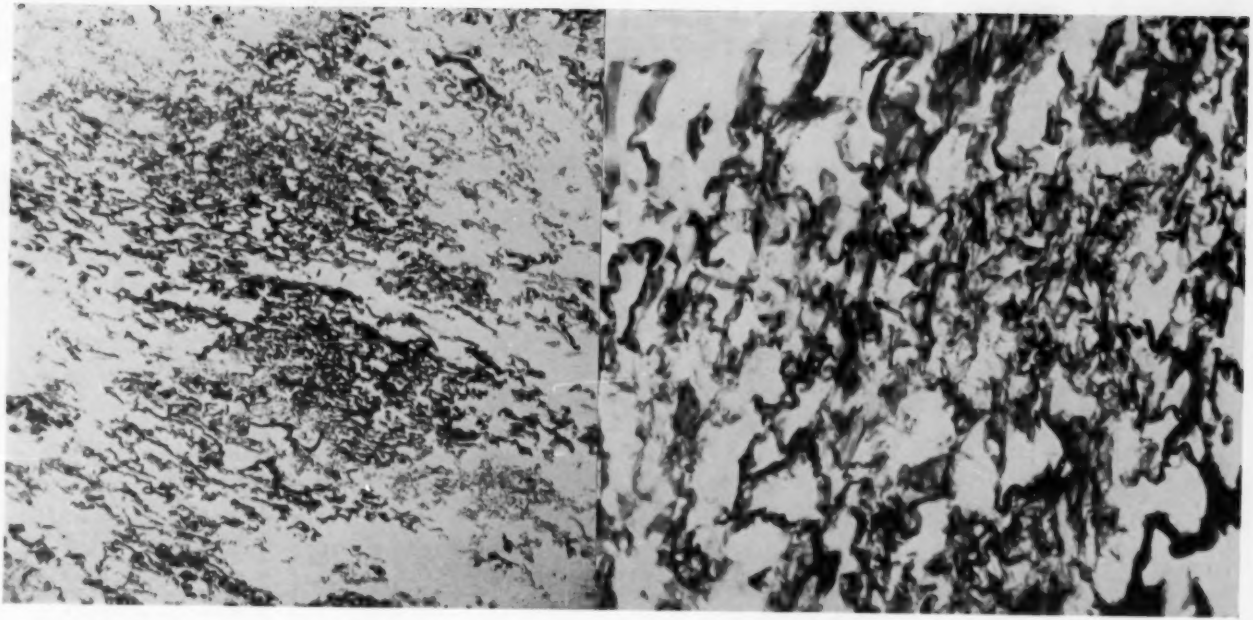

Fig. 2. Cross section of decrystallized submaxillary calculus. PAS stain. A.—Magnification x 137; B.—Magnification x 480.

Fig. 3. Cross section of several decrystallized submaxillary calculi to illustrate tendency to laminations in matrix. A.—PAS stain, magnification x 85; B.—PAS stain, magnification x 85.

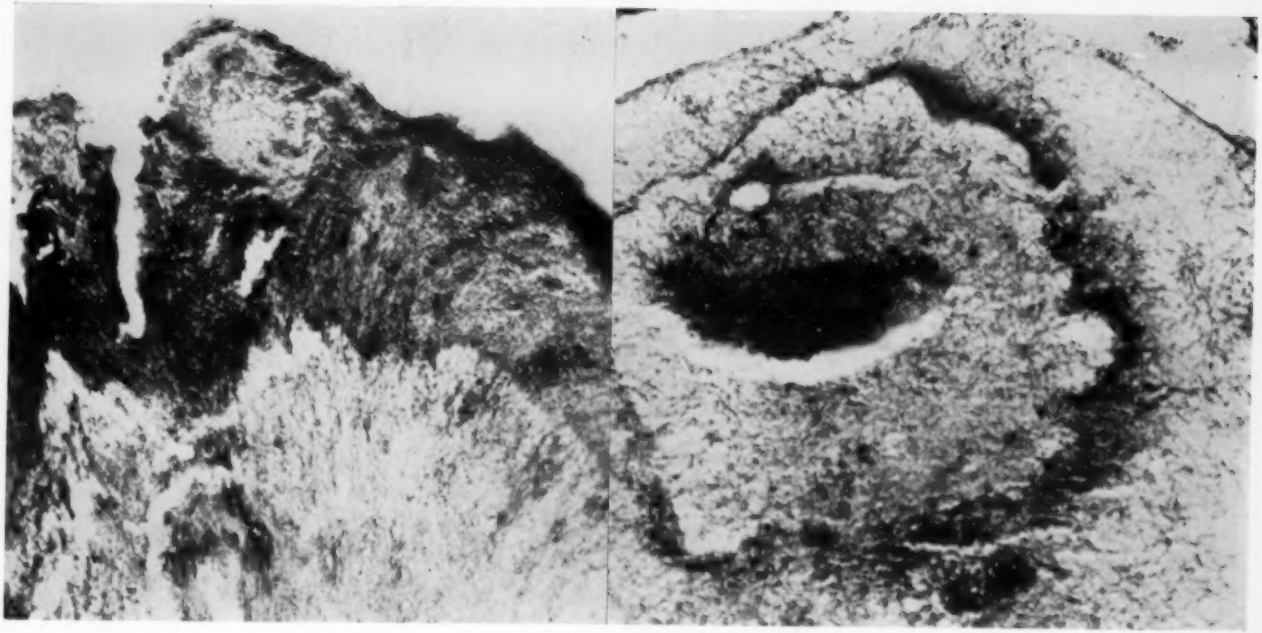

Fig. 3. C.—PAS stain, magnification x 480; D.—Toluidine blue stain, magnification x 480.

matrix with diastase prior to the PAS reaction does not appear to diminish the reaction, and hence glycogen is not a prominent component of salivary matrix. The matrix shows a tendency to fibrillar and intervening amorphus structure quite similar to matrices of urinary calculi. The tendency of the fibrils to form concentric laminations is evident (see Fig. 3), but to a much less degree than in the majority of urinary

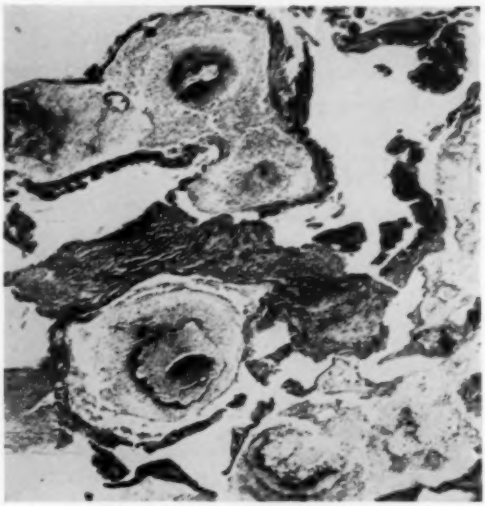

Fig. 4. Cross section of decrystallized submaxillary calculus. Aqueous toluidine blue stain. Magnification x 85.

calculi. No epithelial or blood cells have been seen in the matrices of salivary calculi.

The aqueous toluidine blue gives a uniform orthochromatic (blue) color to the matrix with only rare areas of purplish-red metachromasia (see Fig. 3).

The Sudan black stain gave no indication of lipids within the salivary matrices, and extraction of a single pooled sample of matrix revealed no evidence of alcohol-ether soluble material.

TABLE II.

AMINO ACIDS IDENTIFIED CHROMATOGRAPHICALLY

α Alanine	Glycine
Glutamic Acid	Threonine
Aspartic Acid	Lysine
Serine	Proline
Leucine	(Tryptophane)
Isoleucine	(Tyrosine)
Valine	(Methionine)
Phenylalanine	(Arginine)

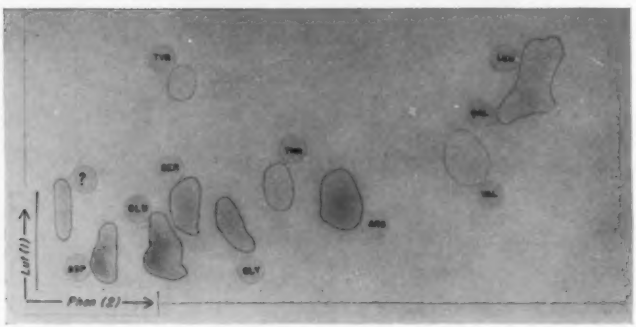

Fig. 5. Two dimensional ascending chromatogram of hydrolyzed calculus matrix in the systems: (1) 2, 6-lutidine:water (65:35 v/v), and (2) Phenol: water (4:1 v/v). Lyophilized matrix hydrolyzed in 6 N HCl for 24 hours at 90° C.

Residue taken up in disodium versenate (0.5 per cent W/v)-isopropanol (10 per cent v/v)-water and centrifuged. The supernatant solution was chromatographed in above systems using Whatman No. 1 paper for 40 to 50 hours. Development by spraying with 0.25 per cent ninhydrin in acetone.

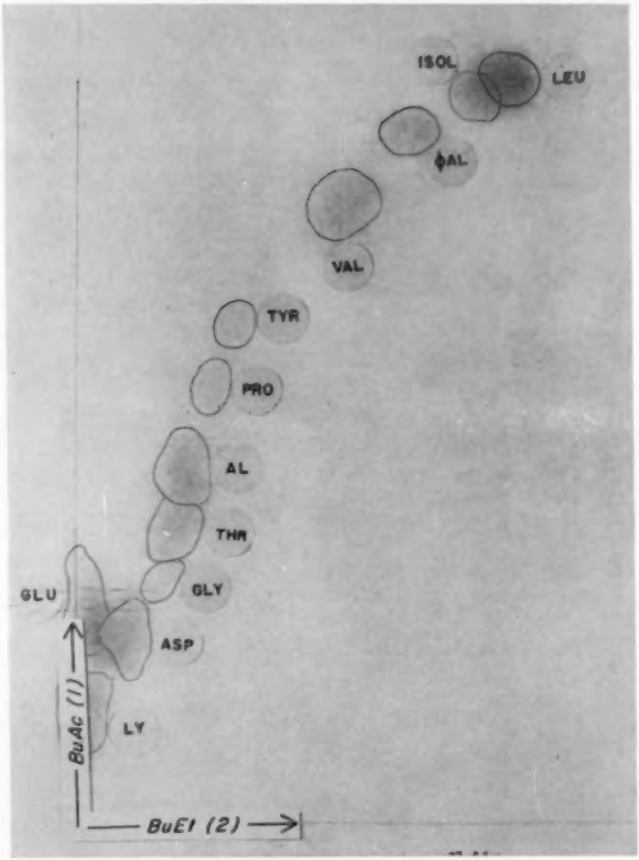

Fig. 6. Two dimensional chromatogram of calculous matrix hydrolyzed as preparation in Fig. 5. Systems were: (1) n-butanol:acetic acid: water (4:1:5 v/v), and (2) n-butanol:ethanol (95 per cent): water (4:1:1 v/v).

TABLE III.

CARBOHYDRATES OF CALCULOUS MATRIX.

Galactose	Hexosamine
Glucose	
Mannose	Deoxypentose
Rhamnose	
Fucose	

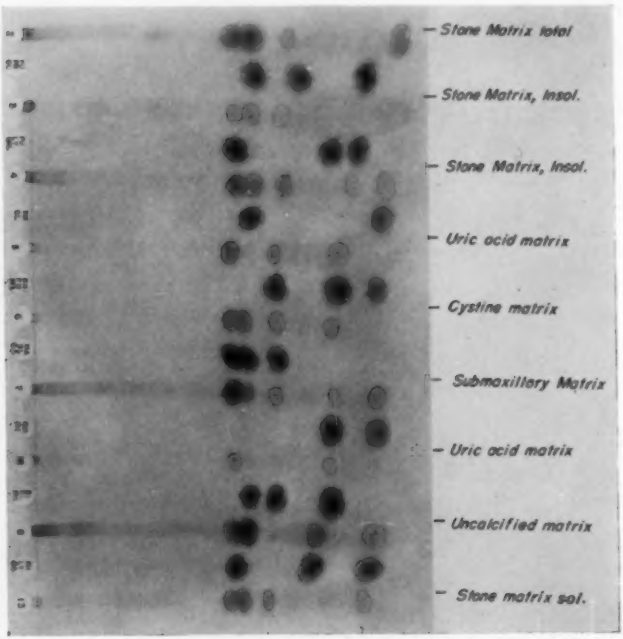

Fig. 7. Comparative chromatogram of carbohydrates from matrices of submaxillary calculi, urinary calculi, and urinary mucoprotein (uromucoid). Lyophilized matrix was subjected to resin hydrolysis for 48 hours (Glegg and Eidinger, Anal. Chem., 26:1365, 1954). Triple ascension was made in n-butanol:acetic acid: water (4:1:5 v/v).

The Von Kossa counterstain of the PAS reactive matrix indicated that most of the sites for silver binding (calcium phosphate deposition) were along the margins of the fibrils.

COMPOSITIONAL STUDIES OF SALIVARY MATRIX.

Matrices from 12 of the larger submaxillary calculi were recovered by lyophilization of the decrystallized calculi.

Amino acid components identified chromatographically in the lyophilized matrix are listed in Table II. Representative two dimensional chromatograms are depicted in Figs. 5 and 6.

The known carbohydrate constituents are listed in Table III. The lyophilized matrix was found to contain five clearly defined carbohydrates: galactose, glucose, mannose, rhamnose and fucose. These carbohydrates have been identified in matrices from both urinary and salivary calculi (see Fig. 7). In addition to these chromatographically identifiable carbohydrates, deoxypentose and hexosamine, have been identified in salivary matrix by previously described methods.[5] Hexuronic acid and "sialic acid" have not been identified in the matrices of salivary or urinary calculi.

SUMMARY.

The striking similarities in microscopic structure and composition of matrix from urinary and salivary calculi have been summarized. Studies of matrix from human fetal bone, gall stones and Mullerian duct calculi have shown distinctly different patterns of carbohydrate and protein content. The electrophoretically identifiable components of saliva[6,7] have many similarities to components of the total nondialyzable solids of urine.[8,9,10,11]

These observations suggest that calculi form as a result of some abnormality of salivary or urinary mucoid. The molecules of mucoid tend to coalesce into a gel and eventually into varying degrees of fibrillar or laminar structures. The organic framework thus forms an architectonic function in the deposition of calcigerous crystals. The extent to which ion binding activity or epitactic stimulation influence crystalliza-

tion remains at present largely a matter of conjecture. There is much evidence that formation of the organic matrix is a prerequisite to calculus formation, and that crystal deposition is a secondary phenomenon. The observation that dental calculus tends to deposit in greatest concentration opposite to the orifices of the submaxillary and parotid ducts suggests a similar mechanism for formation of dental and salivary gland calculi.

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FALSE POSITIVE PERCEPTION DEAFNESS TESTS.*

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Dallas, Tex.

In October, 1953, a communication concerning a necessary revision in our concepts of the hearing tests was read by me before the Section on Ophthalmology and Otolaryngology of the Southern Medical Association.[1] In this paper, after reviewing the tests of hearing utilizing only the at-threshold tests and pointing out their occasional fallibility, especially those involving bone conduction, the added aid to diagnostic accuracy to be found in the above-threshold tests was considered. I said about recruitment, (It is) "a phenomenon appearing in certain cases of perception, or 'nerve' deafness, notably in vascular disturbances of the labyrinth and in presbyacusis. In other such cases it does not appear." In other words, I felt secure in the concept that a case of impaired hearing showing a Weber test, in which the fork was not heard when placed to the forehead or when it lateralized to the better hearing ear, a Rinné test showing air conduction equal to or better than bone conduction, with an audiometric drop in bone conduction and a high tone air conduction loss, or an air conduction loss in the middle tone ranges when coupled with recruitment, was a case with positive indications of cochlear impairment.

I was jolted out of my complacency when the following case came for consideration.

Case No. 1. Mrs. I.C., age 64 years, presented herself on December 29, 1955, complaining of a closed feeling in her left ear and moderate hearing impairment in that ear since the Thanksgiving just passed. The ear disturbance had followed an acute upper respiratory infection. Physical examination was negative, there being only slight bilateral tympanic membrane retraction. No fluid was seen in either middle ear. The results of her hearing tests are shown on her audiogram (see Fig. 1).*

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*All fork tests were done with Riverbank Laboratories calibrated forks. The audiometer used was the Maico E2, and all tests were run in an efficiently sound-treated two-chambered suite.

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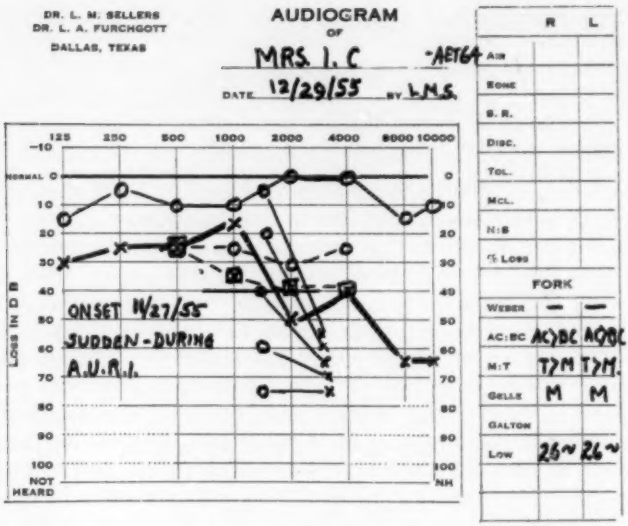


Fig. 1.

Here are present all the criteria for a left cochlear impairment: negative physical findings; the air conduction-bone conduction relationship unchanged and no raising of the lower tone limit with the forks; an audiogram showing a high tone conduction loss; a corresponding drop in bone conduction and the presence of marked recruitment. I was about to tell her the discouraging news that she probably had suffered a post-influenzal perceptive lesion when something caused me to do a thing that I had never done before in a case with such findings; actually I do not know why I did it. Probably it was done to convince her that I had done everything possible before making an unfavorable diagnosis. I catheterized her left eustachian tube. The tube apparently was wide open (the test having been done as always, with binaural auscultation) with no obstruction and no sound of rales. Her face glowed with surprise and happiness as she said, "That's it! I am all right now."

"Psychotherapy;" I thought to myself, "these deafened people always find improvement following any method of treatment;" but I repeated her audiogram. As shown in Fig. 2, it was my turn to be surprised. Two weeks later I repeated her left audiogram, which showed further spontaneous improvement. Then I catheterized her with the same tubal findings; a repeat audiogram showed even further improvement (see Fig. 3).

I considered this to be a freak case and showed copies of the audiograms to several colleagues, who also thought it odd. My having made and saved the copies of these audiograms made possible this report.

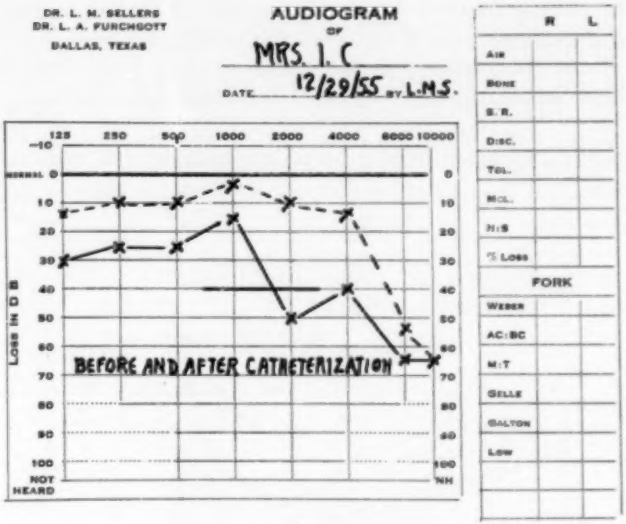
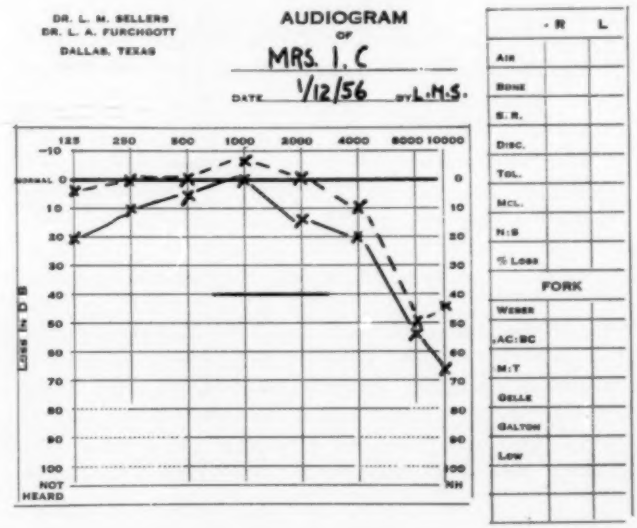

DR. L. M. SELLERS
DR. L. A. FURCHGOTT
DALLAS, TEXAS
AUDIOGRAM
OF
MRS. I. C
DATE 12/29/55 BY L.M.S.
125 250 500 1000 2000 4000 8000 10000
NORMAL 0
LOSS IN D B
BEFORE AND AFTER CATHETERIZATION
NOT HEARD
NH
R L
AIR
BONE
S. R.
DISC.
TOL.
MCL.
N:S
% LOSS
FORK
WEBER
AC:BC
M:T
GELLE
GALTON
LOW

Fig. 2.


DR. L. M. SELLERS
DR. L. A. FURCHGOTT
DALLAS, TEXAS
AUDIOGRAM
OF
MRS. I. C
DATE 1/12/56 BY L.M.S.
125 250 500 1000 2000 4000 8000 10000
NORMAL 0
LOSS IN D B
NOT HEARD
NH
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AIR
BONE
S. R.
DISC.
TOL.
MCL.
N:S
% LOSS
FORK
WEBER
AC:BC
M:T
GELLE
GALTON
LOW

Fig. 3.

In talking about it with Dr. Theodore Walsh, he advanced the speculation that it had been the result of a small droplet of thick mucus lodged in the round window, which was dislodged by the catheterization. This apparently answers the puzzle.

I noticed several cases during the following year that were similar, but they were placed back in the office files as being

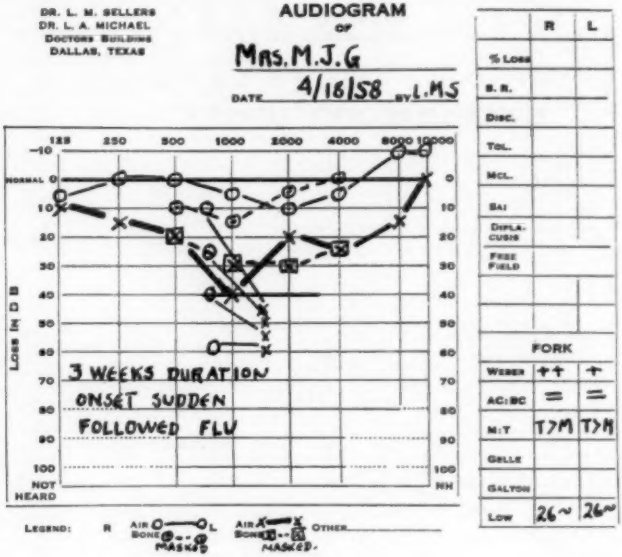


Fig. 4.

merely evidence that unusual cases seem to occur in groups, and so were lost for statistical purposes except as subsequent visits turned them up, because we keep no cross index of diagnoses. Some months ago it occurred to me that these scattered cases may constitute a pattern, and I began keeping a separate record of such cases. Let us consider a few of them.

Case No. 2. Mrs. M.J.G., age 60, was seen first on April 8, 1958. Immediately following a bout with true influenza three weeks previously,

she noticed decreased hearing in her left ear with a feeling as of closure of the ear. Physical examination showed both tympanic membranes to be normal in appearance. There was no visible evidence of fluid in the left middle ear in various head positions, but massage with the Siegel otoscope moved the drum head with a sluggish undulatory motion. With forks the Weber test was referred to the right or sound side; the air conduction equalled the bone conduction bilaterally; the fork was heard better via the tragus than via the mastoid, and the low limit was 26 cps bilaterally. The audiogram (see Fig. 4) showed essentially normal air

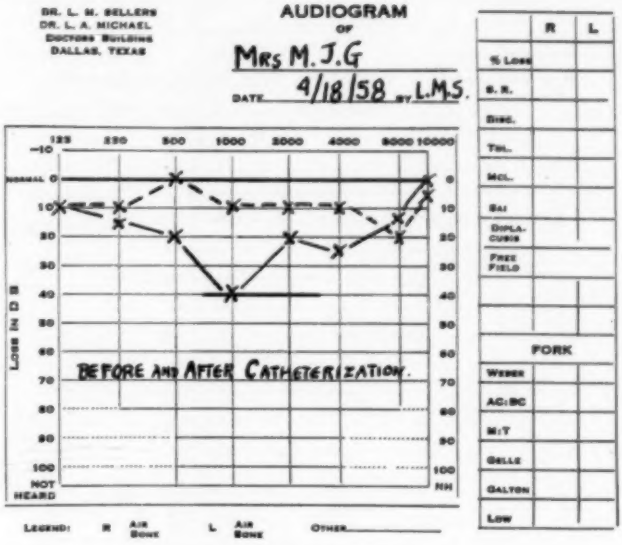


Fig. 5.

and bone conduction in the right ear. There was a mid-range loss in the left ear for both air and bone conduction, the air conduction loss being greatest at 1,000 cps, where she was recruiting markedly. Again all the criteria were present for the diagnosis of cochlear damage.

Catheterization of the left tube revealed moderate occlusion with rales, and after catheterization fluid, with air bubbles, was seen in the left middle ear. Repeat audiogram (see Fig. 5) showed the air conduction to be now essentially normal. She was seen and treated twice more and was dismissed on April 28 with her hearing improvement sustained.

Case No. 3. Miss M.G., age 47, presented herself July 5, 1958. On May 8, she was in an automobile accident with some mild injury to the head. Since then her left ear had bothered her with ringing, roaring and some loss of hearing. No legal action was pending. Physical examination of the ears was normal. The fork tests showed the Weber referred to the left side, undisturbed air conduction-bone conduction and tragus-mastoid

relationships and the low limit to be 26 cps. Her audiogram (see Fig. 6) showed essentially normal air and bone conduction curves for the right ear with a left air conduction low and middle tone moderate loss, there being a 25 db loss at 1,000 cps. The curve rose sharply to normal at 2,000 cps and above normal at 4,000, 8,000, and 10,000 cps. The bone conduction curve for the left ear was 10 to 15 db below the air conduction curve until 4,000 cps where they became identical. At 1,000 cps there was marked recruitment. Here apparently was a clear case of traumatic cochlear damage that I feel sure would have held up in any court of law had a damage suit been pending.

Catheterization of the left tube showed initial occlusion which yielded

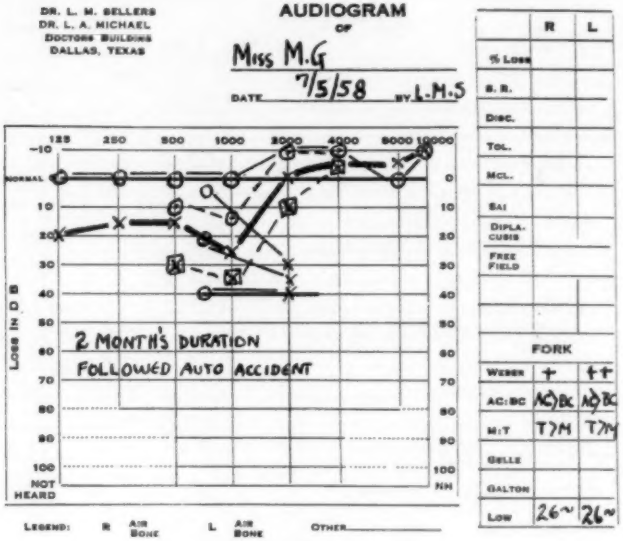
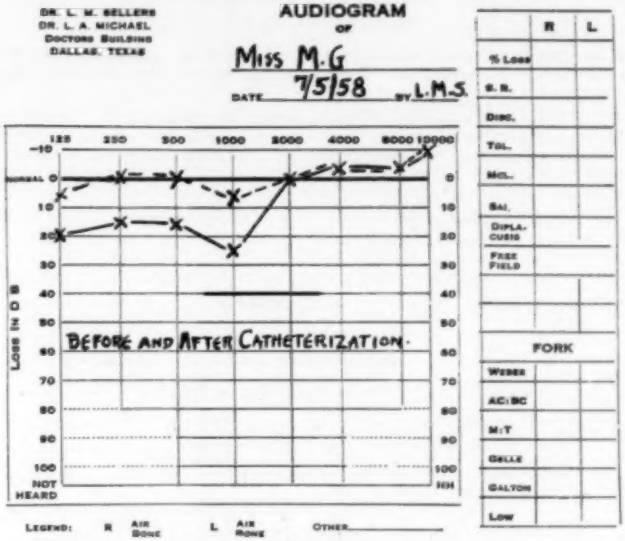


Fig. 6.

to the air current. An audiogram made immediately subsequent to the catheterization presented a normal curve (dash line) as shown in Fig. 7. She was seen again in one week, when a loss less than the original loss was present. This, too, yielded to catheterization. At the third treatment a week later she showed further improvement. This progressive improvement has been maintained by bi-weekly and now tri-weekly sessions.

Case No. 4. Mr. C.F., age 47, was seen first on July 15, 1958. He complained of a loss of hearing in the right ear with a feeling of stoppage. His trouble had existed for three months and had followed an acute upper respiratory infection. Physical examination revealed slight bilateral tympanic membrane retraction without fluid in the middle ears. By the fork tests he referred the Weber to the left (sound) ear; the Rinné test showed the air conduction to be questionably better than the bone conduc-


DR. L. M. SELLERS
DR. L. A. MICHAEL
DOCTORS BUILDING
DALLAS, TEXAS
AUDIOGRAM
OF
Miss M.G
DATE 7/5/58 BY L.M.S.
125 250 500 1000 2000 4000 8000 10000
LOSS IN D B
NORMAL
NOT HEARD
BEFORE AND AFTER CATHETERIZATION.
LEGEND: R AIR BONE L AIR BONE OTHER
R L
% LOSS
S. R.
DISC.
TOL.
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FREE FIELD
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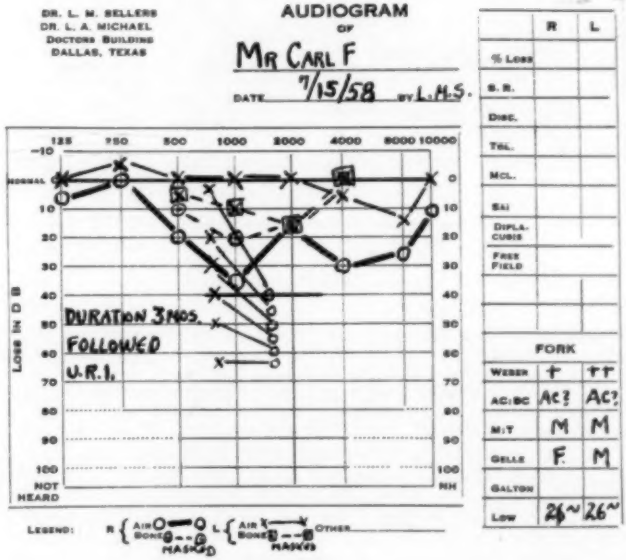
Fig. 7.


DR. L. M. SELLERS
DR. L. A. MICHAEL
DOCTORS BUILDING
DALLAS, TEXAS
AUDIOGRAM
OF
MR CARL F
DATE 7/15/58 BY L.M.S.
125 250 500 1000 2000 4000 8000 10000
LOSS IN D B
NORMAL
NOT HEARD
DURATION 3 MOS.
FOLLOWED
U.R.I.
LEGEND: R AIR BONE MASKED L AIR BONE MASKED OTHER
R L
% LOSS
S. R.
DISC.
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MCL.
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DIPLACUSIS
FREE FIELD
FORK
WEBER T TT
AC:BC AC? AC?
M:T M M
GELLE F M
GALTON
LOW 24 26

Fig. 8.

tion, hearing by mastoid was greater than by tragus bilaterally, the Gelle test was positive for fixation of the left stapes, and the low limit was 26 cps bilaterally.

The audiogram (see Fig. 8) was completely normal for the left ear, but revealed a mid-tone range loss by air conduction in the right ear, with the greatest loss at 1,000 cps, where there was marked recruitment. The bone conduction curve was lowered definitely, but was not lower than the air conduction curve.

Catheterization of the right tube disclosed slight occlusion, which opened under moderately light pressure and without rales, but without relief of

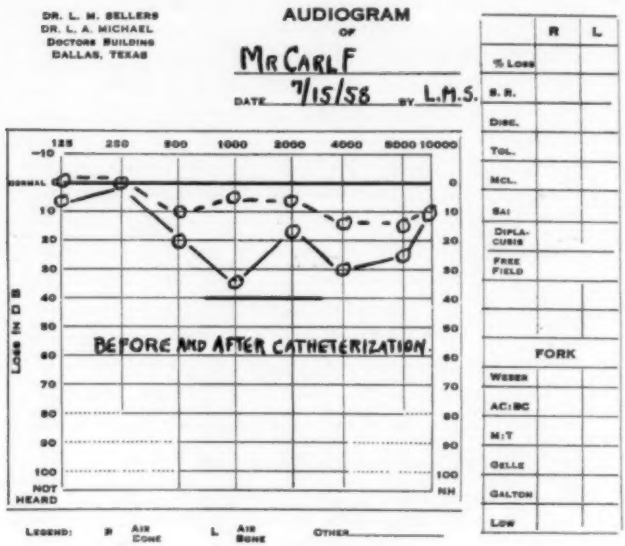

Fig. 9.

his feeling of fullness and deafness. Recalling Dr. Walsh's opinion that this picture could be produced by heavy mucus clinging to the round window, I repeated the catheterization, using increased pressure until the drum head was distended and very slight vertigo occurred. A repeat audiogram (see Fig. 9) showed a marked rise of the air conduction curve to almost the normal level.

On July 15, there was a recurrence of his symptoms. The tube was found to be occluded, and rales were heard when the occlusion yielded, but without relief. The drum head was incised, and catheterization was repeated. This time a profuse amount of straw colored fluid was blown into the canal. On July 24 the drum was healed, and his hearing was essentially normal.

On August 18 there was a recurrent tubal occlusion, and on September

12 incision again yielded fluid, but much less than on the first occasion. His progress since has been uneventful.

Case No. 5. Mr. W.M.R., age 60, came to us first on July 15, 1958, the same day on which we first saw Case No. 4. There was fullness with diminished hearing in the left ear of two and one-half months' duration and with no attributable cause. He felt better, and he heard better when lying on his left side. There was marked retraction of the right tympanic membrane. The left tympanic membrane was moderately retracted and a scant amount of fluid, whose level shifted with change of head position, was present.

The fork tests showed the Weber to be without lateralization, the Rinné demonstrated air conduction to be greater than bone conduction; hearing

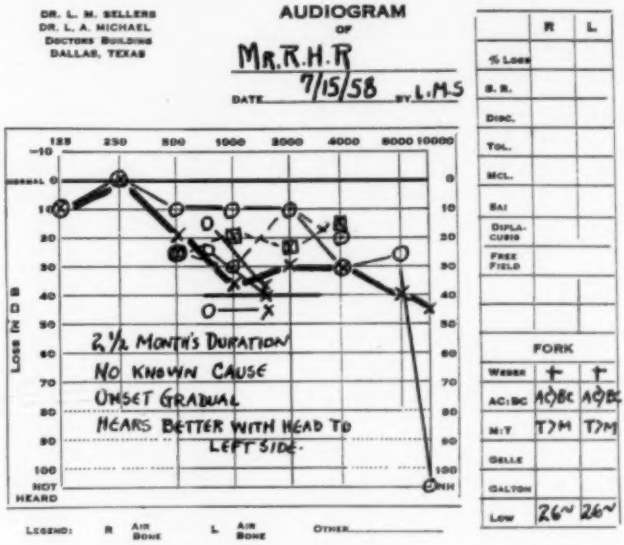

Fig. 10.

by tragus was better than by mastoid, and the low limit in both ears was 26 cps. The right ear showed audiometrically (see Fig. 10) a high tone air conduction loss commensurate with his age; the left ear a middle and high tone loss. There was bilateral lowering of the bone conduction threshold curve greater in the right ear at 1,000 cps. In the left ear there was marked recruitment at 1,000 cps. Catheterization of the left tube revealed moderate occlusion, which yielded to gentle pressure with the appearance of bubbles in the middle ear fluid. A repeat air conduction audiogram (see Fig. 11) showed marked improvement in the middle range, the hearing level now equalling that of the right ear. Myringotomy, followed again by catheterization, produced a profuse dis-

charge of yellow fluid. The improvement in his hearing level remained the same and he was discharged on July 21. He has been seen subsequently concerning another otolaryngological problem, and his hearing was the same as on July 21. In this case the history and the clinical and audiological pictures substantiate Dr. Walsh's proposal that the cochlear syndrome in these cases is produced by occlusion of the round window. It will be recalled that the patient stated in his history that turning onto the left side (which by gravity would tend to clear the round window) partially relieved his hearing disability.

Case No. 6. Mrs. A.Y., age 54, was seen on August 30, 1958, because of severe progressive hearing loss and tinnitus in the left ear. Her complaint

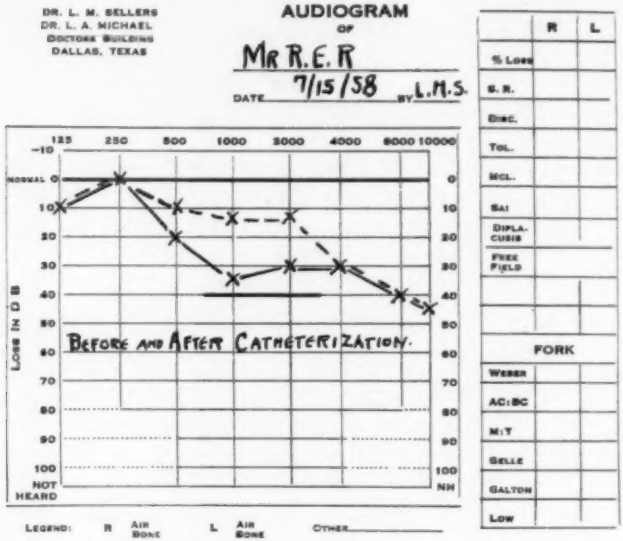

Fig. 11.

had been present for six weeks, and had followd an infection of the external auditory canal. Her voice was characteristic of severe perception deafness, wooden in tone, high pitched and very loud.

The canals, tympanic membranes and middle ears were quite usual in appearance. By fork tests the Weber was heard in neither ear, the Rinné test showed the air conduction to be greater than bone conduction, hearing by tragus better than by mastoid, and the lower limit to be 26 cps, all bilaterally (see Fig. 12).

The audiometric air conduction curve of the right ear showed a slight tip downward to the right with marked notching to 60 db at 8,000 cps. The bone conduction curve approximated that of the air conduction curve. The left air conduction curve showed marked lowering through-

out with a marked drop to the right, with notching at 8,000 cps that carried the curve out of hearing limits. The bone conduction curve, although higher than the air conduction curve, was lowered markedly but rose to the right. Because of the marked lowering of the air conduction curve of the left ear loudness balancing was not feasible. Fortunately, this omission remedied itself later.

Catheterization of the left tube revealed moderate occlusion, which yielded to pressure and without rales. There was marked hearing improvement (see Fig. 13), relief of the feeling of fullness and complete loss of the wooden quality of the voice.

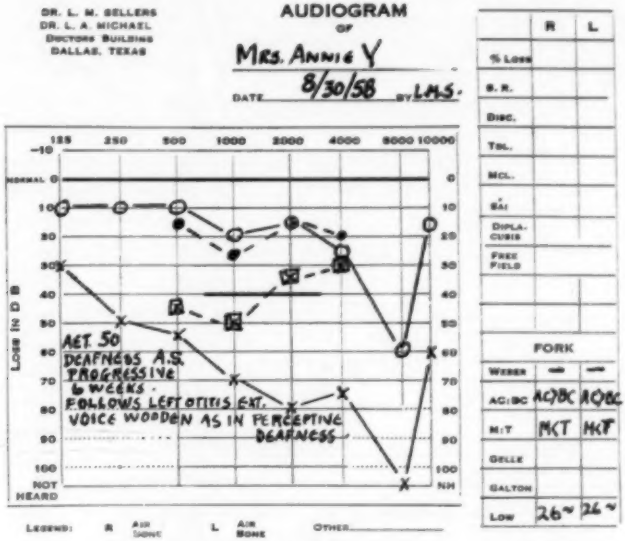

Fig. 12.

Three days later there was a marked spontaneous improvement which was increased by catheterization (see Fig. 13).

On September 8 there was a moderate recurrence of her symptoms and findings, as shown in Fig. 14, but this time loudness balance at 4,000 cps was feasible, and it revealed very marked recruitment. Catheterization immediately brought her air conduction curve up to approximately that of her right ear. Her improvement has been steady, and at the time of this writing there has been no further recurrence.

Case No. 7. Mr. F.S., age 42, was first seen on June 3, 1957, complaining of hearing loss with tinnitus in the left ear for three months. Its onset occurred during an attack of acute rhinitis. There was no vertigo. With the fork tests the Weber lateralized to the right, the Rinné was normal in both ears, and the low limit bilaterally was 26 cps (see Fig. 15).

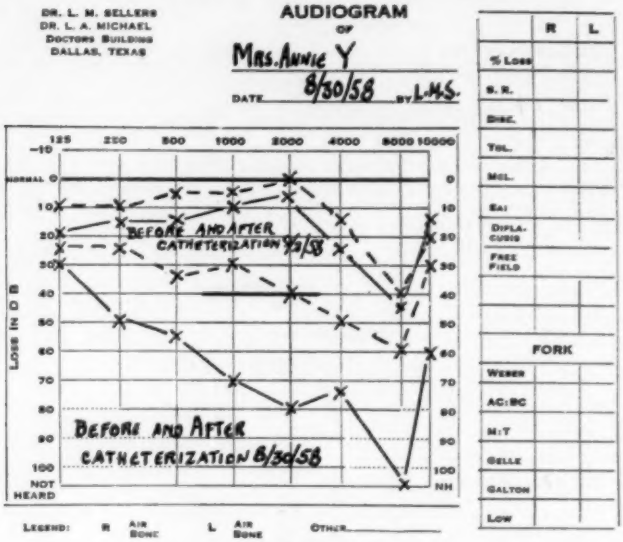
DR. L. M. SELLERS
DR. L. A. MICHAEL
DOCTORS BUILDING
DALLAS, TEXAS
AUDIOGRAM
OF
MRS. ANNIE Y
DATE 8/30/58 BY L.M.S.
BEFORE AND AFTER CATHETERIZATION 8/30/58
BEFORE AND AFTER CATHETERIZATION 8/30/58
LOSS IN D B
NOT HEARD
NH
LEGEND: R AIR BONE L AIR BONE OTHER
R L
% LOSS
S. R.
DISC.
TOL.
MCL.
SAI
DIPLACUSIS
FREE FIELD
FORK
WEBER
AC:BC
M:T
GELLE
GALTON
LOW

Fig. 13.

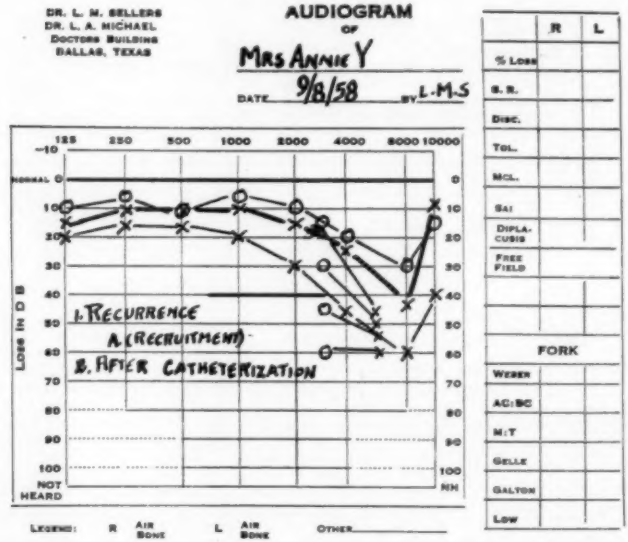
DR. L. M. SELLERS
DR. L. A. MICHAEL
DOCTORS BUILDING
DALLAS, TEXAS
AUDIOGRAM
OF
MRS ANNIE Y
DATE 9/8/58 BY L.M.S
1. RECURRENCE
A. (RECRUITMENT)
B. AFTER CATHETERIZATION
LOSS IN D B
NOT HEARD
NH
LEGEND: R AIR BONE L AIR BONE OTHER
R L
% LOSS
S. R.
DISC.
TOL.
MCL.
SAI
DIPLACUSIS
FREE FIELD
FORK
WEBER
AC:BC
M:T
GELLE
GALTON
LOW

Fig. 14.

Audiometrically the right air conduction curve remained at the zero line until 2,000 cps, after which point it dropped continuously. The bone conduction curve was depressed and roughly followed the air conduction curve. The left air conduction curve showed a level depression at 30 db until 2,000 cps, after which it roughly followed the right air conduction curve at a 5 to 15 db lower level. The left bone conduction curve was lower than the air conduction curve and roughly followed it. Recruitment was present in the left ear at 1,000 cps. The tube was widely patent, and no improvement in hearing followed catheterization. A final diagnosis of cochlear impairment was made, and he was placed on vitamin

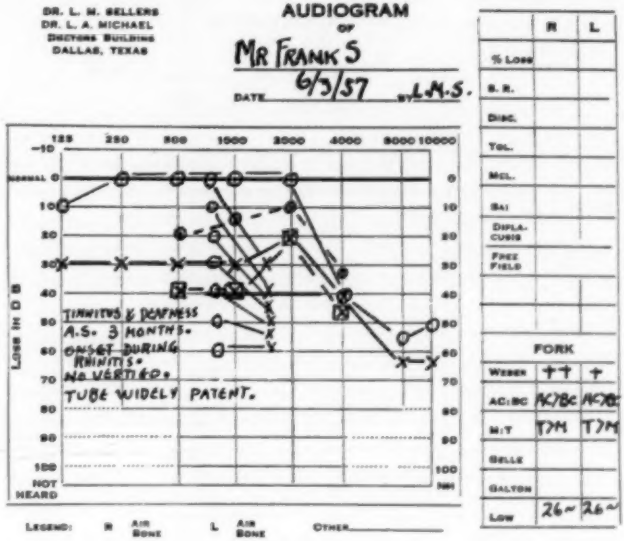

Fig. 15.

vasodilators. His air conduction curve fluctuated upward and downward, as shown by the dash lines on Fig. 16 for July 1, 1957, and March 4, 1958.

On August 26, 1958, he came to the office and stated that his "ear had popped open yesterday" and that he felt completely relieved. His air conduction at this time is indicated by the heavy solid line in Fig. 16. This episode could have resulted only from something dislodging itself from some critical area in the middle ear, and I can imagine no site other than the round window for this.

The cases that we have discussed here are of great importance to all of us in our practice.

It has become a habit lately for otologists to look down

their noses at catheterization of the eustachian tube, so much so that it has become almost a lost art. Those who speak of it may do so at the cost of being considered in the twilight zone of practice. This is understandable. For all too many men in otology in the recent past it was the *vade mecum* of their practice, and it was used whether indicated or not. A reaction following Newton's third law was inevitable. I am afraid

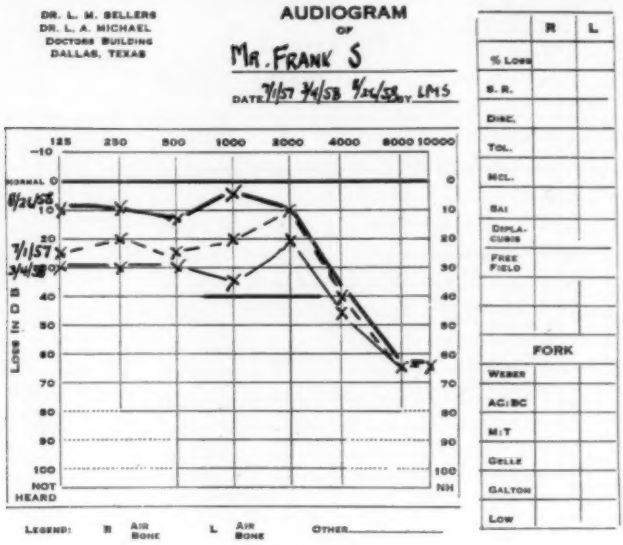

Fig. 16.

that I have been almost as guilty in this respect as anyone. I dread to think of how many poor souls who presented to me the pictures that I have presented to you today (with the exception of those who showed physically the presence of fluid in the middle ear) have been told by me that their difficulty was only possibly amenable to treatment by oral or parenteral medication; and who, when they received no benefit, went to a less "informed" and less "scientific" medico than myself and found relief; or far worse, those who trusted me com-

pletely and went on without relief to which they were entitled by Almighty God. This thought of misplaced trust is a grave one to bear, even though the error occurred while the best of medical thought and practice of the time was being utilized. It is not enough that we did our best. Our best simply was not good enough.

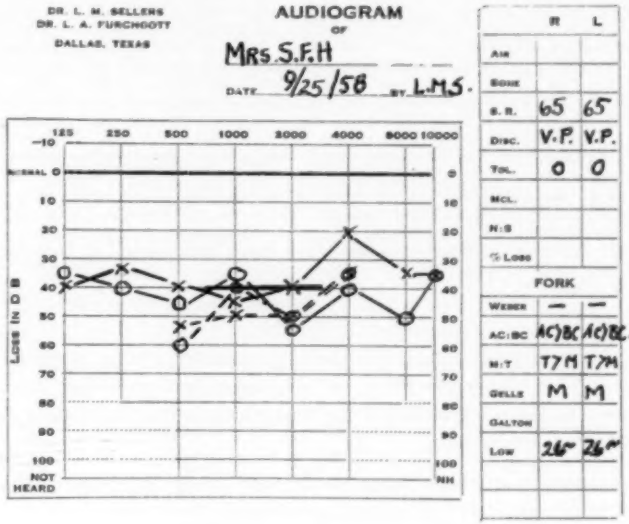

Fig. 17.

It now has become the practice in our office to catheterize every case in which unilateral cochlear impairment has been diagnosed. This, I am sure, will account for the increasing frequency with which these cases presenting false positive perception deafness tests are being uncovered; and we are looking with only a slightly less jaundiced eye on cases diagnosed as bilateral cochlear impairment.

In spite of all that we have just said, less than a fortnight after the foregoing portion of this communication was completed the next case presented itself.

Case No. 8. Mrs. S.F.H., age 42, was referred on September 25, 1958, by the Vocational Rehabilitation Division of the Texas State Education Agency for whatever help we could give her, as she was about to lose her job because of marked deafness. This deafness had been present since the age of 13 years and was of unknown origin. Her hearing tests, as shown in Fig. 17, were typical for perception deafness—the flattened curve for both air and bone conduction with marked raising of both thresholds. The fork tests and the speech reception tests were in complete agreement with the pure tone audiogram. Only an occasional word could be distinguished at 55 db. Raising or lowering the intensity by 5 db lost even that meager hearing gain; however, she was an excellent (self-taught) lip reader. Catheterization revealed both tubes to be patent, and it did not improve the hearing. A diagnosis of perception deafness was made, and she was told as gently as possible the gloomy outlook. Fortunately, a friend as a last resort took her to a hearing aid dealer who, after making an audiogram, which later proved to be quite similar to the one we had made, empirically tried a bone conduction hearing aid. It worked. He happened to be a man of high ethics in his calling and he called me by phone to tell me what had happened. I had him return her to me for a check of my own findings. These were as they were at the first examination with the following exception: The speech reception threshold, using the bone conductor to the right mastoid, was found to be at 25 db with excellent discrimination, as she heard practically all words on a spondee list of 50 words. With the bone conductor over the left mastoid the left ear showed no improvement in speech reception. We simply have no explanation to offer for this bizarre case. It remains a challenge and a warning.

SUMMARY.

A short series of cases has been presented, giving audiological evidence of perception or cochlear deafness, but which proved to have been caused by disturbances in the middle ear conductive mechanism.

Warning that these cases occur much more frequently than past experience would suggest is offered.

In conclusion, we suggest the possibility that cases Numbered 3, 4 and 5, with middle ear fluid, demonstrated physically which simulated cochlear impairment, but with hearing loss most notable in the middle tone range, may suggest a pattern as an aid in diagnosing the presence of fluid in the middle ear when the fluid is not to be seen on examination.

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INJURIES AND SEQUELAE ASSOCIATED WITH ENDOTRACHEAL ANESTHESIA.*

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The advantages of the endotracheal method of anesthesia are well known. There has, however, been relatively little written about the disadvantages of this procedure which has come to be so eminently useful to us. An ever increasing sense of awareness of the possible sequelae of endotracheal anesthesia has been manifest during the past decade. Our attention has been particularly stimulated by Flagg,[1] Mintz and Adriani,[2] Moss,[3] Baron and Kohlmoos,[4] and Holinger and Johnston.[5] The majority of the work published on this problem has consisted of reports of over one hundred cases of laryngeal granulomata following endotracheal anesthesia.

This presentation will deal with the causes, the suggestions for prevention of intubation accidents and sequelae, and a description of the more serious untoward reactions seen during the past twelve years.

The undesirable effects of intubation should be divided into those caused by, *1.* inexpert use of the laryngoscope; *2.* traumatic passage of the endotracheal tube; *3.* the presence of the tube itself in the air passages.

Many of the sequelae following this method of anesthetic administration are due to faulty knowledge or faulty application of a few basic principles. Probably the most frequent is that of the patient's position during laryngoscopy. The usual "anesthetist's position" is that of the lambda of the head forming a pivot with the surface of the table, the point of the chin being directed vertically. The upper teeth form a fulcrum between the battery handle and spatula of the laryngoscope. Using this fulcrum the base of the tongue and

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epiglottis are pried anteriorward allowing a view of the posterior larynx. By placing the left thigh across the end of the operating table and resting the patient's head on the thigh, one has achieved a position with which the larynx can be quite easily exposed by merely lifting the base of the tongue and epiglottis.

A frequent error in technique is the failure to identify the structures across which the tip of the laryngoscope passes. Observation of this error led Moss[3] to state, "ploughing up the pharynx with a laryngoscope in an attempt to dig out an epiglottis from a pool of bloody mucus is one of the least inspiring sights of modern anesthesiology." The anatomy should be recognized step by step beginning with the palate and proceeding in turn to the posterior pharyngeal wall, base of the tongue, tip of the epiglottis, arytenoid eminences and finally the vocal cords. During this progression down the pharynx, secretions are best aspirated by a straight rigid tube rather than a soft catheter as the latter is more difficult to manage with one hand.

Inexpert use of the laryngoscope has caused a variety of mouth, pharyngeal and laryngeal injuries. Myerson[6] states, "All of us have seen broken dentures, loosened or avulsed teeth, and contusions or lacerations in the region of the ascending ramus of the mandible, the tonsillar pillars, the tongue base, the epiglottis or the pharyngeal walls, and last, but by no means least, the structures of the larynx."

Full appreciation of the delicate structures with which the endotracheal tube is in contact will demand of the anesthetist great care and gentleness in the introduction of the tube. Many of us have seen lacerations within the nose due to the excessive force applied through a narrow nasal airway. Misdirection of the tube so that it does not pass down the lower meatus often leads to the belief that there is inadequate space to accommodate it. Much of the bleeding frequently seen during nasal intubation is due to abrading the adenoid, and is usually of little significance. Blind intubation, both nasaliy and orally, should not be practiced except in circumstances where it is inadvisable or impossible to laryngoscope the patient. Blind and traumatic passage of the endotracheal tube is cred-

ited by some observers as being a factor in the formation of laryngeal granulomata and other sequelae.[6]

Case 1. Seen in consultation in 1953. The patient, a young lady, was sent to the Ear, Nose and Throat department for suture of a laceration sustained two hours previously during direct laryngoscopy for the purpose of endotracheal anesthesia. The laceration extended into the floor of the mouth on the left side as far anteriorly as the second molar, inferiorly to the angle of the mandible, and posteriorly along the base of the tongue to the level of the epiglottis. There was very little bleeding. The wound had not been discovered until the operation had been completed, and the patient complained of pain in the area. After repair she made an uneventful recovery.

COMMENT.

Apparently the anesthetist was struggling with a patient insufficiently relaxed. Failure to identify the passing anatomy resulted in the technician's becoming lost. It is difficult to understand how enough force could be applied to cause such a laceration. A smaller tear of the anterior tonsillar pillar caused by the collapse of the head of the operating table while the laryngoscope was in place, has been observed.

Case 2. T.H., a colored man, aged 52, was scheduled on May 26, 1955, for removal of an extremely large goitre. Persistent attempts to intubate were unsuccessful, and the operation was postponed because of moderate respiratory difficulty. Breathing became progressively worse during the day, and in the early evening the patient was in a state of extreme respiratory obstruction. An emergency tracheotomy was performed at the bedside. The larynx and trachea were found to be displaced 4 cm. to the left of the midline, causing the tracheal incision to be made beneath the anterior border of the left sternocleidomastoid muscle. Aeration improved immediately. At midnight, during a coughing spell, considerable bleeding was initiated. A large open thyroid vein was found and tied. Mirror laryngoscopy, the following day, revealed total obscurity of the interior of the larynx by edema and a large hematoma involving the entire left side of the larynx. There were several areas of ecchymosis in the pharynx and hypopharynx. Laryngeal respiration gradually returned, and on June 15, 1955, a thyroidectomy was done. Final mirror laryngoscopy a month after the accident revealed no trace of the original injuries.

COMMENT.

This demonstrates the difficulty encountered by a change in anatomic relationships and the resultant injuries caused by too vigorous attempts to force a tube into place. It also demonstrates the remarkable healing power of these tissues and their ability to withstand serious injury and yet return to a state of near normality. Training, experience and judg-

ment are worth just as much in this field as in any other specialty.

Case 3. A white male was scheduled for removal of a lump from the right side of his neck. The surgeon's pre-operative workup, including mirror laryngoscopy, failed to reveal any abnormality of the larynx or pharynx. Endotracheal anesthesia was chosen. During the laryngoscopy, extensive bleeding from the hypopharynx occurred. A fellow anesthesiologist was immediately called to examine the patient. He was of the impression that there was a tumor above the right vocal cord. Early in the afternoon a laryngologist was asked to see the patient. Mirror laryngoscopy revealed extensive edema of the right side of the larynx and pyriform sinus. The entire right side of the hypopharynx was obliterated by the swelling. Direct laryngoscopy immediately set off rather brisk bleeding, and the procedure was discontinued. Ten days later much of the swelling had subsided, and direct laryngoscopy revealed a large fungating tumor of the right pyriform sinus, which was biopsied. The pathologist reported squamous cell carcinoma. Ten days following this a laryngectomy, right hypopharyngectomy and right radical neck dissection were performed. The tumor was highly vascularized and extremely friable.

COMMENT.

The difficulties encountered during intubation procedures through abnormal or pathological anatomy should be kept in mind. The haste often associated with intubating under pentothal adds to the problems of the anesthetist. Laryngological consultation in neck tumors should be requested more frequently.

Although the immediate untoward results associated with endotracheal anesthesia are due to trauma, this is not entirely true of those seen hours or weeks later. Laryngeal edema is seen frequently and is thought to be due to one, or a combination of factors such as an oversize tube, excessive trauma during the passage of the tube through the larynx, friction of the tube against the structures surrounding it, and chemical irritation initiated by the solution used to sterilize the tube.[7]

Necrosis of tracheal mucosa has been reported as due to pressure of the inflated cuff. Periods of anesthesia in excess of six hours are thought to be necessary in producing sufficient ischemia to bring about the necrosis.[8]

The vocal cord polyps occasionally seen are, in all probability, due to the organization of a small submucosal hemorrhage originating during the passage or withdrawal of the

tube. Excessive voice use during the first few post-anesthetic days may also be a factor.

Hematoma within the larynx may vary from slight submucosal hemorrhages along the cord edges to massive collections large enough to obstruct the airway. The former are usually due to indelicate introduction of the endotracheal tube, and the latter to unusual and severe trauma, inflicted by the tip of the laryngoscope.

The most frequently reported late sequelae of endotracheal anesthesia is the now thoroughly familiar laryngeal granuloma.

Case 4. On February 3, 1953, Mrs. B.F. sought relief of her hoarse voice, wheezing respiration, and shortness of breath. She had a pelvic operation of long duration three months previously. There was no report of the introduction of the endotracheal tube as having been traumatic. Immediately on recovery from the anesthetic the patient noticed that her voice was slightly husky, and after four days it became hoarse. She was told that this would soon disappear, and on discharge from the hospital several days later it had improved. She resumed her usual vocal habits, admitting more than moderate voice use, and in approximately three weeks was again fairly hoarse. Often after coughing, phonation would be completely lost for a few seconds. There was a constant sensation of phlegm in the larynx. Wheezing and moderate shortness of breath gradually became manifest.

Mirror examination revealed bilateral 4 mm., rounded, grayish-purple, pedunculated masses attached to the vocal cords just anterior to the vocal processes. They could be blown above the cords by the air blast of coughing, and when in the inferior position were almost completely covered during phonation. On February 4, 1953, direct laryngoscopic removal was performed, using topical anesthesia of pontocaine 1 per cent. Microscopic study revealed chronic inflammatory tissue. The voice returned to normal, and there had been no recurrence up to the time of the patient's change of residence to another city several months later.

COMMENT.

It is felt that vocal abuse could have been a prominent factor in the formation of these lesions. Several days of voice rest following endotracheal anesthesia, particularly in patients who are bent toward excessive vocalization, may be helpful in preventing this sequela.

The etiology of laryngeal granulomata has been the subject of much speculation, as exemplified by the following list of factors thought to have a bearing on their formation:

1. The size of the tube.[7,12]
2. The solution in which the tube was sterilized.[7]
3. Friction between the larynx and the tube.[11,12]
4. Blind intubation.[6]
5. Febrile upper respiratory infections.[12]
6. Elastic pressure of the tight fitting tube.[7]
7. Chemical composition of the tube.[7]
8. Construction of the advancing tip of the tube.[7]
9. Technique of introduction.[7,10]
10. The curve of the Magill tube.[8,9]
11. Face down position during anesthesia.[12]
12. Duration of the anesthetic.[12]
13. Post-intubation vocal abuse.[12]

TABLE I.

INJURIES AND SEQUELAE SEEN DURING PAST TWELVE YEARS.

CHILDREN	ADULTS
1. Laryngeal edema necessitating tracheotomy.	1. Severe laceration and separation of base of tongue from lateral pharyngeal wall.
2. Laceration of anterior pillar of tonsil.	2. Pinching of upper lip with laceration.
3. Avulsion of teeth.	3. Broken denture.
4. Broken teeth.	4. Broken teeth.
5. Broken orthodontic appliances.	5. Avulsion of teeth.
6. Hematoma of vocal cord.	6. Laceration of edentulous gingival margin.
7. Ecchymosis of pharynx.	7. Laceration of turbinate.
8. Tracheitis.	8. Laceration of adenoid and turbinate.
9. Laceration of adenoid.	9. Contusion of mandible.
10. Tear of aryepiglottic fold.	10. Laryngeal granulomata, bilateral.
11. Laceration of lip.	11. Tracheitis.
12. Contusion of gingiva.	12. Laceration of adenoid.
	13. Hematoma of vocal cord, extensive.
	14. Ecchymosis of pharynx.
	15. Vocal cord polyp.
	16. Massive edema and hematoma of larynx.
	17. Nasal hemorrhage.
	18. Severe laceration of a pyriform sinus carcinoma.
	19. Laceration of aryepiglottic fold.
	20. Functional dysphonia.

It is generally agreed that there must be trauma to the thin mucoperichondrium covering the medial aspect of the tip of the arytenoid cartilage. The mere passage of a tube

through the larynx, even if traumatic, does not necessarily initiate the formation of granuloma. Multiple bronchoscopies or the leaving of a bronchoscope within the airway for many hours has not been reported as causative.

O'Dwyer self-retaining endolaryngeal tubes were used extensively during the years in which diphtheria was prevalent. No case of laryngeal granuloma has been reported following their use. These tubes moved up and down with the larynx during coughing, gagging and swallowing, and thus imparted no frictional trauma to the vocal cords. They did produce untoward reactions but not granulomata. Myerson[13] stated, "The O'Dwyer tubes, when used in too large a size, caused subglottic edema first. If permitted to stay in place a long time, ulceration, perichondritis, necrosis, distortion, and tracheotomy followed. When a proper size tube was used, the patient had no permanent sequelae." Clerf[14] routinely used the largest tube that could be easily introduced, since a smaller size was too readily coughed out.

The use of rubber core molds, or plastic obturators in the treatment of laryngeal injuries has produced none of these lesions; yet these appliances are of large size, and are permitted to remain within the larynx for weeks and months. They move with the larynx and create no friction.

It has been suggested that the curve of the Magill tube may play a prominent part in the etiology. Dwyer, Kronenberg, and Saklad,[8] believe that this is a most important factor in the production of traumatic effects of endotracheal anesthesia, and have substituted a double curved tube.

One of our basic principles in laryngeal treatment has been enforcement of periods of voice rest. After denuding the vocal process by the endotracheal tube, may not the trauma of vocal abuse play a prominent role in the final development of a granuloma? Barton[12] feels that voice rest would reduce the incidence of this lesion.

CONCLUSIONS.

It is felt that the more serious injuries associated with endotracheal anesthesia stem from the violation of basic prin-

ciples of laryngoscopy, and a failure to appreciate the delicate anatomic structures involved. Training in laryngoscopy should be stressed among anesthesiologists. Some of the causative factors in late sequelae are inherent faults of the endotracheal method of anesthesia. It is speculated that removal of the mucosa covering the junction of the membranous vocal cord and the arytenoid are not, in themselves, responsible for granuloma formation, but that there is needed in addition the hammer and anvil action of vocal abuse to forge these lesions.

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CORRECTION.

In the paper "A Method of Removing a Nasal Hemangiopericytoma" by W. Burbank Woodson, published in The Laryngoscope for April, 1959, on page 447, the last paragraph should read ". . . . . a very large growth measuring 2.5x1.7x1.3 cm. was removed . . . . ."

ADENOIDS, TONSILS, AND TWO DECADES OF CHEMO-ANTIBIOTIC THERAPY.*†

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As is customary in the preparation of any medical report, some of the literature dealing with the tonsil and adenoid problem in recent years was reviewed. Boies[1] (1948) noted that from "1942 to 1946 inclusive, there has been an average of 100 papers per year concerning tonsils, listed in the *Quarterly Cumulative Index*; and this in spite of the war years when medical literature was less voluminous than in ordinary times."

Interest in the subject has not diminished in the past ten years. Various aspects of the matter, such as pre-operative medication (Rendall); pre-operative management (Jonkees); the use of vitamin K (King); the internists' viewpoint (Badger); general aspects (Eley); the relation of hearing (Hoople); stress on more complete removal (Meltzer); the anatomy and histology of Rosenmueller's fossa (Reeves and Brill); the complications and their management (Work), and mortalities and morbidity (Cummings), were all noted by Wishart,[2] et al. in a review of the literature (1954). Newer concepts concerning the preventive influence of tonsillo-adenoidectomy on the common cold are reviewed by Hollender,[3] who concludes: "No causal relationship between the pharyngeal lymphoid structures and the common cold (or other respiratory tract diseases) can logically be considered unless the existing tonsillar disease falls in the category of so-called focal chronic tonsillitis." The specific role and problems of the adenoid are discussed by Senturia,[4] who stresses the importance of the complete inspection of the nasopharynx during operation. McGovern[5] reviews the advantages of endotracheal

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anesthesia and notes that the introduction of the Salk vaccine has probably altered the importance of the much-debated association of tonsillectomy and poliomyelitis.

Johnston and Watkins[6] (1954) felt that the decision to remove tonsils and adenoids has been too conservative for the past 30 years. They studied 598 children following tonsil and adenoid removal. One hundred twelve were between the ages of 1 and 4 years, 331 were between the ages of 5 and 7 years, and 155 were between the ages of 8 and 14 years. They noted a reduction in ear infections, decrease in abdominal pain referable to chronic infection, and fewer attacks of bronchitis. Patients with sinusitis (13 out of 28), and asthma (three out of 22), showed least improvement after operation. The period or duration of follow-up studies was not clear. They concluded that tonsillectomy and adenoidectomy remains a highly-beneficial procedure in the properly selected cases.

There appeared to be a surprising lack of papers related specifically to the use of antimicrobials and tonsil-adenoidectomy. Orzac[7] studied 500 children 2 to 12 years old, subjected to tonsil and adenoidectomy in 1954 and 1955. Pre-operative Adrenosem, one mg. three times a day, and a mixture of benzathine penicillin and triple sulfonamides, one teaspoonful three times a day, was given for seven days. At operation, one cc. of Adrenosem and one cc. procaine penicillin were administered intramuscularly, and repeated two hours after completion of surgery. On discharge from the hospital, benzathine penicillin-triple sulfonamide mixture with codein, one teaspoonful four times a day, was continued for six days. Three hundred patients, as controls, received no medication before or after surgery.

"Records were kept on each patient concerning the occurrence of *1.* primary and secondary hemorrhage; *2.* postoperative infections, as determined by clinical examination and evidence of undue elevation of temperature, and *3.* postoperative pain, as measured by the number of telephone calls from parents and questioning of the parent and child in the postoperative period.

"Primary hemorrhage occurred in 1.7 per cent, and secondary hemorrhage in 1 per cent of the 300 control patients, but in none of the 500 children who received preoperative and postoperative medical treatment.

"Postoperative infections retarded convalescence in 13 (4.4 per cent) of the 300 unmedicated controls, but in only seven (1.4 per cent) of the 500 treated children.

"Three of the treated patients had catarrhal acute otitis media, three pharyngitis with adenitis, and one had bronchopneumonia. Viral gastroenteritis developed after operation in another patient in this group but was not counted a true postoperative complication, since three family contacts had become ill with the disease the day before the child was operated upon.

"In the unmedicated group otitis media occurred in seven patients, the acute catarrhal form in six, and purulent acute in one. Two had bronchopneumonia, three had pharyngitis with cervical adenitis, and one had cystitis.

"Postoperative pain severe enough to warrant one or more phone calls from the parents for additional treatment was three times as frequent for the controls as for the medicated patients."

Riggs[8] indicated that pre- and postoperative penicillin should be considered, but not given unless definitely indicated for protection, and that "sulfonamides and other antibiotics pre- and postoperatively may be justified."

A brief but concise report, with special reference to the allergic implications on respiratory symptoms and the influence of tonsil and adenoidectomy is that of Clein[9] (1952), who summarized: "The operation for removal of tonsils and adenoids should be performed for adequate indications in any child whom the physician feels will be benefited by this procedure. Most failures are due to the fact that similar symptoms of allergic etiology are overlooked. Correct diagnosis of the allergic disease, followed by thorough specific treatment will often prevent this operation, as well as certain common complications." He also refers to the important study of Kaiser[10] (1940), who wrote on the significance of the tonsils in the development of the child.

The stimulus for the following comments evolved from the clinical impression gained during the past few years that many children are being referred for tonsil and adenoidectomy at a very early age. The history invariably reveals one or more of the following: *1.* Recurrent attacks of tonsillitis and adenoiditis; *2.* repeated attacks of tonsil and adenoiditis with associated otitis media; *3.* clinical indications of sensitivity to one or more chemo-antibiotic agents; *4.* decreased interval of freedom from upper respiratory tract infections involving the tonsils and adenoids; *5.* definite resistance to the beneficial effects of chemo-antibiotic and medical therapy; *6.* frustrated parents or physicians, faced with the recurrently ill

or irritable child, necessary drug bills, medical fees, sleepless nights, and restraint of adult social activity. All patients are physician referred.

Specifically, this study was undertaken to: *1.* determine, if possible, whether these observations were unique on a local basis or indications of a national trend; *2.* compare the age levels for the removal of tonsils and adenoids before the introduction of chemo-antibiotics with the present; *3.* determine, if possible, the role of chemo-antibiotic agents at the time of tonsil and adenoidectomy; *4.* obtain some idea as to the present indications for the operation; and *5.* observe the effect of pre- and postoperative treatment with chemo-antibiotics. The results of a study of the latter (No. 5) will form the basis of a future report.

After much thought and investigation, it was found that, due to the method of keeping physicians' and hospital records, introduction of new hospitals, and many other factors, no reliable data (local) in the period prior to 1936 (prechemo-antibiotic period) could be assembled for comparison purposes.

This discovery led to the development of a short sampling questionnaire, which was mailed to at least two or more active otolaryngologists, aged 50 years or over, in each State of the Union. Complete returns numbered 101. One of these was eliminated to simplify results. The findings are summarized as follows:

Question No. 1.

Is it your impression that in general there has been a change in the age level for tonsil and adenoid operations during the past ten years (as compared to the period 1928 through 1938)?

TABLE I.

Forty-six replied NO; Fifty-four replied YES.

Degree of Change	Lowered	Increased
1 year	7	11
2 years	11	7
3 years	12	6
	30	24

None of the above were supported by statistical studies but were rendered as clinical impressions.

A more definite evaluation was submitted by Simonton[11] comparing the age and number of operations for the years 1935 and 1957.

1935—Age	Number	1957—Age	Number
2	13	2	26
3	10	3	30
4	19	4	41
5	15	5	47
6	19	6	46
7	22	7	36
8	13	8	21
	111		247

Question No. 2.

Number in order of your impression the three most frequent precipitating factors or indications leading to tonsil and adenoidectomy in the age zero to 10 years.

The answers were as follows: *First,* multiple attacks of primary tonsil and adenoiditis, 80; *second,* repeated attacks of early otitis media, 62; *third,* obstruction to breathing, 30; *fourth,* reduced hearing (not acute), 25; *fifth,* repeated attacks of resistant otitis media, 13; and 4 each for persistant cervical noditis, resistance to the benefits of chemo-antibiotics, common cold, and focal factors. Seven listed a single miscellaneous condition.

Question No. 3.

Do you prescribe chemo-antibiotics routinely?

TABLE II.

Before Operation		After Operation
80	None	62
9	1 day	16
3	2 days	8
8	3 days or longer	6
	7 days or longer	8

Question No. 4.

List the two complications noted most frequently within the first ten days after tonsil and adenoidectomy.

Bleeding within the first 24 hours after operation was listed by 40, and after four days or during the slough period by 30. Thirteen replies specified the adenoid region. Infection (not classified and presumably of the postoperative sites) was the second most frequently noted by eight. Among the postoperative symptoms and conditions noted were sore throat, otolgia, vomiting, fever, dehydration, anorexia, otitis media, upper respiratory-tract infection, and worried mothers, in the order mentioned. None indicated the relation of antimicrobials to the complications or symptoms.

The results of the questionnaire are suggestive of a slight trend to an earlier age for the operation. To determine any possible correlation at the local level, we then reviewed the ages of all patients having undergone tonsil and adenoid operations and performed by Operator A at one hospital during the seven-year period 1952-1958, inclusive. The findings are noted in the following:

TABLE III.

Age	1952	1953	1954	1955	1956	1957	1958	Total
0-1			1	1	1	1	2	6
1	1	1		1	5	1	3	12
2	24	20	13	18	25	20	31	151
3	40	23	34	16	24	32	39	208
4	32	29	32	15	35	45	42	230
5	38	35	36	15	19	37	50	230
6	19	42	21	15	31	45	38	211
	154	150	137	81	140	181	205	1048

TABLE IV.

Age	1952	1953	1954	1955	1956	1957	1958	Total
7	5	20	21	12	26	19	28	131
8	5	7	10	5	14	16	15	72
9	6	3	5	3	13	13	16	59
10	1	3	4	3	8	5	5	29
11	1	3	1		7	6	5	23
12		2	1	1	3	1	1	9
	18	38	42	24	71	60	70	323

In Table III, it will be observed that the youngest patients, less than one year old, totaled 6; in the 1-to-2-year-old period,

TABLE V.

Age	1952	1953	1954	1955	1956	1957	1958	Total
13		2	3		5	1	4	15
14			3				2	5
15			1	1	2		3	7
16	1	1			1			3
17							1	1
18			1		2	3	2	8
19	1	1			1			3
	2	4	8	1	11	4	12	42

TABLE VI.

Age	1952	1953	1954	1955	1956	1957	1958	Total
0-6	154	150	137	81	140	181	205	1048
7-12	18	38	42	24	71	60	70	323
13-19	2	4	8	1	11	4	12	42
20 or older	6	10	11	3	10	9	7	56
Total	180	202	198	109	232	254	294	1469

there were 12; in the 2-to-3-year-old period there were 151; and the majority, in about equal distribution, in the 3-through-6-year age group.

Table No. 4 shows a marked decrease in the number of operations performed after age 7 years. The ratio of operations for both tonsil and adenoidectomy or tonsillectomy alone, is about the same for the age group from 13 years through 19 years when compared to all ages from 20 years and above.

From these results, it can be noted that the majority of tonsil and adenoid operations are performed in the age period from 2 through 7 years, and the period between 3 years and 6 years represents the greatest concentration period for the operation in this series.

FACTORS CONTRIBUTING TO THE OPERATION AT AN EARLY AGE.

Indications.

It was of interest to note that the indications in our group followed very closely those of the questionnaire group. Every patient was physician referred, and presumably a medical therapy failure. A close review of the group (254) under-

going surgery in 1957 showed that primary tonsil and adenoiditis was responsible for the operation in 69 per cent; tonsil and adenoiditis with history of colds and otitis media and with or without hearing impairment, 26 per cent; and all other causes 5 per cent. All of the patients under age 12 years had persistent cervical noditis, and the majority had a history of decreased effectiveness of chemo-antibiotic therapy with each succeeding attack.

Anesthesia.

The introduction of endotracheal tubing by the experienced anesthetist team, contributing to a safer method of anesthesia administration, is, in our opinion, a major factor of importance in permitting a safe tonsil and adenoid operation at any age, but particularly in young children. This encourages the referring physician and the parents to seek or consent to an early operation.

Control of Bleeding.

This, in our opinion, is (next to safe anesthesia) the most important factor. Multiple factors are responsible for excessive bleeding. With careful preparation, this phase can be minimized by: *1.* having the patient at his best physical state; *2.* minimal degree of infectious state possible; *3.* control of bleeding at the time of surgery; and *4.* the use of chemo-antibiotic therapy pre- and postoperatively, as indicated on an individual basis.

Rhoads, et al.[12] stated that "prolonged antibiotic therapy not followed by extirpation of the tonsils may be serious, even if the micro-organisms that were the orginal objects of the antibacterial attack are temporarily eliminated." Their summary is worth noting:

> Blood cultures taken just after tonsillectomy were positive in 28.3 per cent of a group of 68 patients who received no antibiotic therapy prior to tonsillectomy. The incidence of bacteria was reduced to 5.9 per cent in a group of 20 subjects who received penicillin in a daily dose of 600,000 to 800,000 units intramuscularly for four to ten days prior to tonsillectomy. The incidence of bacteria in a group of 29 patients who received 600,000 to 800,000 unit doses (half this dose for children) of procaine penicillin 12 to 18 hours and one hour prior to tonsillectomy or in a small group (seven) of patients receiving 900,000 to 1,200,000 units orally daily for

five to seven days prior to operation was not reduced below that of the control group. Beta hemolytic streptococci were obtained from blood cultures four times, pneumococci once, Alpha hemolytic streptococci (green forming) or Gamma anhemolytic streptococci 28 times, and a combination of Beta hemolytic streptococci and Gamma anhemolytic streptococci twice. The need for several days preoperative treatment with penicillin to prevent post-tonsillectomy bacteremia is obvious.

In the control series of 68 patients who had no antibiotic treatment immediately preceding tonsillectomy, Beta hemolytic streptococci were present in 57.4 per cent of the cultures of the excised tonsils, although these micro-organisms were found in only 28.26 per cent of throat cultures taken just before the operation. Among patients receiving penicillin in single doses only the day before and the day of tonsillectomy, 31.03 per cent had Beta hemolytic streptococci in the excised tonsils, although these micro-organisms were not present in the throat cultures taken just before tonsillectomy. Beta hemolytic streptococci were found only once in the cultures from the excised tonsils of persons receiving penicillin intramuscularly each day for four to ten days prior to tonsillectomy. Most of the gram-positive micro-organisms except micrococci and Gaffkya tetragenus were greatly reduced in number by penicillin administered intramuscularly, but gram-positive micro-organisms, such as Klebsiella pneumoniae, Aerobacter aerogenes, and Escherichia coli, were found in increased numbers in cultures from the throats and excised tonsils of these subjects.

SUMMARY.

Accurate statistics for comparison of adenotonsillectomy age levels before and after the introduction of chemo-antibiotics (1936) are not readily available.

Chemotherapy and antibiotics apparently have not eliminated the need for the operation below the age of seven years.

The *use in early infancy* of chemo-antibiotic therapy to help control the systematic spread of infection and reduce the complications of cold and tonsil-adenoid infections.

The apparent ineffectiveness of chemo-antibiotics when used repeatedly.

The apparent lack of immunity of many young children to recurring attacks of tonsil-adenoid infection with or without colds.

The value of pre- and postoperative anti-microbials.

Improved techniques for safe anesthesia, public knowledge of medical facts, and public acceptance of hospitalization care.

The above, collectively, are some of the factors contributing to adenotonsillectomy at an early age.

1200 E. Broad St.

Medical definitions:

Antibiotic—1. Destructive of life. 2. Pertaining to or characterized by antibiosis. The term antibiotics is given to antibacterial substances of biologic origin. They are derived from bacteria (pyocyanase, pyocanin, tyrothricin); from actinomyces (actinomycetin, actinomycin, streptothricin, streptomycin); from molds and fungi (penicillin, citrinin, gliotoxin, fumigatin, fumigacin, claviformin, flavicin, flavicidin and others); from natural substances other than micro-organisms (lysozyme, chlorellin, canavalin, allicin).

Chemotherapy—The treatment of disease by administering chemicals which affect the causative organism unfavorably but do not injure the patient.

COURS DE TECHNIQUE AUDIOMETRIQUE.

The Faculty of Medicine, Paris France, will hold a Course in Audiometry, October 12-18, 1959, under the chairmanship of Prof. M. Aubry of the Clinic of Oto-Rhino-Laryngology. The tentative program includes lectures on the scientific fundamentals, the technique of audiometry, discussions of the results and problems of interpretation, and round table discussions. Professor Aubry will be assisted by MM. les Professeurs Agreges J. J. Debain and R. Maduro; les Doctears P. Aboulker, J. Bouche, P. Clerc, H. Henrot, R. Maspetion, J. Pialoux, J. Bouchet, M. Burgeat, L. Chevance; P.-L. Klotz, P. Robert, B. Vallancien; Mme. S. Borel-Maisonny; M. le Professeur A. Didier; MM. R. Chocholle, J.-E. Fournier, R. Lehmann and A. Molles. The complete program will be ready in June, 1959. For further details write Secretariet: Service Oto-Rhino-Laryngologique Pavillon Isambert, Hospital Lariboisiere. 2 Rue Ambrose-Pare, Paris 10°), France.

THE LYMPHOID TISSUE OF THE NASOPHARYNX.*†

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Although the nasopharynx is no longer the neglected structure it was charged to be a few decades ago, current knowledge concerning the role of its lymphoid tissue remains vague. This intricate type of tissue is not restricted to the postnasal space, however, masses of various proportions being scattered normally in other areas of the body. Because of the structural similarity of these widely strewn tissue masses, the idea of regarding their total aggregation as a single functioning organ is not without a logical basis. Here we are concerned only with the lymphoid tissue of the nasopharynx (see Fig. 1). The relationship of this tissue to its own space, to other parts of Waldeyer's ring (see Fig. 2), and to the body as a whole provides a problem of vast clinical significance.

In recent years scholarly articles have been contributed on the subject. Research has ranged from fundamental histopathologic studies to precise observations on the effects of hormonal drugs. Through these diversified investigations new ramifications are constantly being exposed. Although it is realized that many aspects of the overall problem of lymphoid tissue still need to be clarified, understanding of some of its phases has been removed from the realm of speculation.

DEFINITION AND FUNCTION.

Lymphoid tissue cannot be defined logically without reference to the lymphoid organs. According to Clark,[1] these consist of mere irregular accumulations of lymphoid cells, termed lymphoid infiltrations, and are found in mucous mem-

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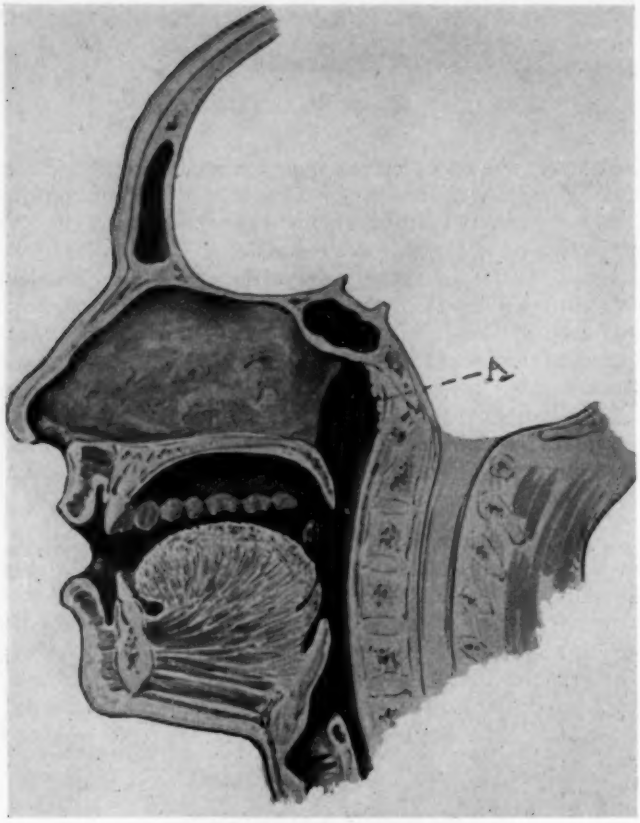

Fig. 1. The nasopharynx; A, lymphoid tissue structure.

branes, especially along the intestinal tract and the air passages in the lungs. The lymphoid organs are closely associated with the lymph vascular system.

According to Drinker and Yoffey,[2] lymphoid tissue consists of a mass of free cells, the vast majority of them lymphocytes of various sizes, together with a supporting framework of reticulum cells and of fibrous elastic and sometimes muscular

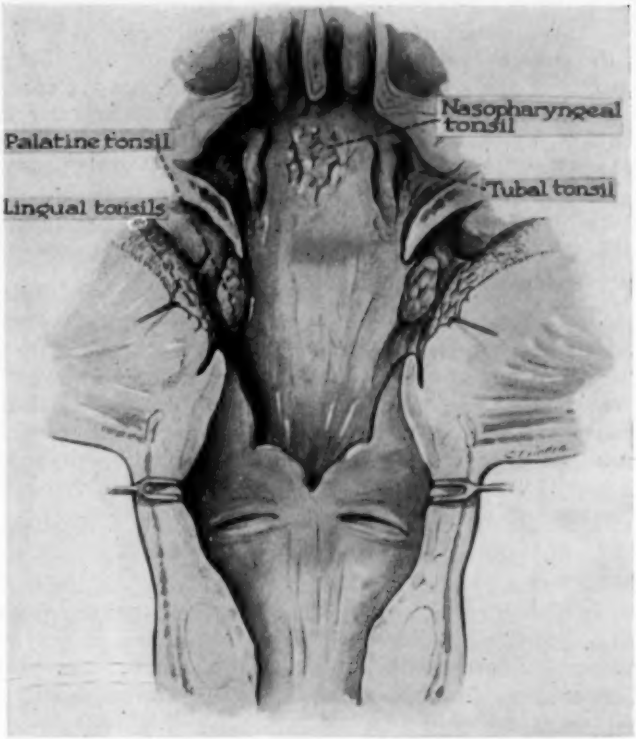

Fig. 2. Lymphatic ring of Waldeyer; the pharyngeal tonsil and the other lymphatic structures.

elements. In their recent book, Yoffey and Courtice[2a] defined lymphoid tissue as "any tissue consisting predominantly of lymphocytes, such as lymph nodes, tonsils, appendix, and intestinal nodules . . . the lymphocytes in lymphoid tissue are usually intermingled with other elements, to which they may be either functionally or genetically related."

As concerns the lymphatic system, it may be described as a type of circulatory system, an accessory to the venous system—

a filtering and defense mechanism.[2] The lymph structures of the nasopharynx probably have the same functions as those of the general lymphatic system.[3] Hochfilzer[4] held that the function of the lymphatic system of the oropharynx depends on the whole lymphatic plexus. Like others, Sharp[5] assumed that the function of the postnasal lymphoid tissue was protective in nature. He stated: "Developing, as it does, in common with lymphoid tissue elsewhere in the body, it is ideally situated to act as a protective filter to the respiratory passages during early childhood, when infection is frequent and development immunity low."

Lymphoid tissue is said to be concerned in antibody formation. Although knowledge of the role of the cells of the reticuloendothelial system in this regard is indeed meager, it is reasonable to accept the conclusion of Topley and Wilson,[6] "that the tissue cells themselves are capable of producing antibodies in the fully formed state." On the one hand, according to Semenov,[7] tonsils and adenoids produce lymphocytes, and on the other, they destroy them.

For the purpose of clarification, Yoffey and Courtice[2a] drew an analogy between the function of lymphoid tissue and that of certain other organs. They contended that the production of lymphocytes is as much a normal function of lymphoid tissue as is the formation of digestive juices a function of the intestinal mucosa, or the formation of bile, a function of the liver.

Yoffey and Courtice[2a] acknowledged that "difficult, as it may be to understand the role of lymphoid tissue in disease, the problem becomes even more perplexing when we consider the function of lymphoid tissue in health." In the latter state, the following functions have been listed by these investigators: *1.* lymphocyte production; *2.* metabolism and transport of protein and fat; *3.* vitamin storage; *4.* elaboration of internal secretion; *5.* destruction of red cells; *6.* the provision of nucleoprotein and other substances as adjuvants to cell growth.[2a]

GROWTH OF LYMPHOID TISSUE.

Knowledge concerning the growth of lymphoid tissue has

been derived from animal experimentation. Although the main problems entail the role of nutrition and the hormones, some factors of lesser importance also warrant consideration.

Nutrition. According to White,[8] an adequate caloric intake is essential for the growth of lymphoid tissue. Other workers (Settles, Jackson), likewise, have observed that lymphoid tissue is reduced in amount during starvation (inanition) or malnutrition, and increased above normal by rich feeding, especially in children.

The protein content of the diet is an important factor in relation to lymphoid tissue growth. Proof of this is afforded by restricting the intake of proteins or essential protein constituents.[9] If an increase in total serum protein takes place, it is due essentially to an increase in the globulin fraction. Lymphoid tissue has a high requirement for pyridoxine. Involution of this tissue has been observed in animals on a pyridoxine-deficient diet.[10]

Hormones. The concept of a hormonal-lymphoid relationship is supported by the fact that the administration of certain hormonal substances alters the size of the tissue mass by reducing or increasing it. Evidence suggests, moreover, that the secretion of adrenocortical hormones, controlled by the pituitary adrenotropic hormone, is the normal mechanism regulating lymphoid tissue mass. In brief, the pituitary adrenal apparatus influences the size and development of lymphoid tissue.[8]

Histologic changes in lymphoid tissue render it possible to determine the effect of adrenocortical hormones. Although the effect seems to be prompt, various stimuli are able to augment the secretion of adrenotropin. As the rate of secretion of this substance is increased, the production of adrenocortical steroids is increased. The result is that the steroid hormones influence in particular the processes which yield to its stimulation. The accelerated release cannot occur, however, in the absence of adrenotropic hormone.[8]

Criscenti[11] investigated the relationship between the endocrine glands and Waldeyer's lymphatic ring. By controlled studies on dogs he was able to show a reduction in size of the

tonsillar structures with Cortisone and ACTH. He realized, however, that the effect would be reversed when the hormones were discontinued; but he held out hope that through some future device the effect would be rendered permanent.

Observations like these, according to White,[7] serve to explain why the various physiological changes affected by pituitary-adrenocortical secretion may be seen, to a qualitatively similar extent, in response to a wide variety of agents and in diverse circumstances. White contended that this endocrine mechanism is undoubtedly the physiologic basis for the so-called accidental involution of lymphoid tissue seen in the "alarm reaction" of Selye,[12] inasmuch as stress, whether physical or chemical, is one of the potent activators of pituitary-adrenocortical secretion.

Miscellaneous Factors. Lymphoid tissue mass can be influenced also by factors not related to the pituitary-adrenocortical mechanism. For example, alterations in mass size have been produced in the hypophysectomized or adrenalectomized animal by exposure to radiation, mustard gas and the like, or by the injection of certain toxic substances.[2a]

Although infection in lymphoid tissue usually affects the size of its mass, this change, contrary to general belief, is not invariable. Nasopharyngeal tissue undergoes cellular arrangement in response to many systemic diseases. The role of adrenal insufficiency in producing delayed involution of lymphoid tissue has already been mentioned. Allergy and some of the major blood disorders have been described prominantly in this connection.[13] As concerns allergy, clinical experience points to it as one of the common causes of lymphoid tissue enlargement. After seeing so much recurrence even following exact surgical intervention, Meltzer[14] concluded that allergy undoubtedly plays a cardinal role. In his opinion, this contention is strongly supported by the number of patients in whom improvement is seen following allergic management. Although my own observations from the standpoint of etiology are in strict accord with those of Meltzer, I have not shared his encouraging attitude concerning the allergic aspect. In my experience, allergy as a factor is not usually considered until the nasopharyngeal process has developed

with fixed changes. Because of this, treatment is not uniformly successful.

HYPERPLASIA OF NASOPHARYNGEAL LYMPHOID TISSUE.

In a study conducted 15 years ago, Szanto and I[14] pointed out that of the various concepts concerning the development and involution of nasopharyngeal lymphoid tissue, the one more commonly accepted is the so-called "cranio-caudel development" of Waldeyer's ring. Since a complete description of this aspect must necessarily embrace all of the structures of Waldeyer's ring, it is not within the scope of this present writing. Suffice, that development of the nasopharyngeal tonsil begins after birth and attains its peak between the second and third years of life. At puberty, involution of this structure, generally but not always, approaches completion. Under abnormal circumstances, however, especially in the presence of allergy or chronic infection, hypertrophy of lymphoid mass usually persists. This pathologic change is the replacement for atrophy which, under normal circumstances, would develop and progress throughout life.

Infancy and Childhood. Hypertrophy* (or hyperplasia) of nasopharyngeal lymphoid tissue in infancy or childhood is usually referred to as adenoiditis (see Fig. 3). This tissue normally is of greater amount in the child than in the adult, decreasing steadily in size as age advances. Because of its situation and the type of mucous membrane covering it, the adenoid plays a prominent role in respiratory physiology. If this lymphoid mass becomes enlarged, crowding the postnasal vault, it loses its defensive powers and provides a culture-bed for infection which readily spreads to contiguous structures. Respiratory and otologic complications are inevitable consequences.

Older Persons. It is generally assumed that at puberty involution of lymphoid tissues nearly always approaches com-

*According to Warren ("Pathology," Edited by W. D. Anderson, 3rd Ed., The C. V. Mosby Company, St. Louis, 1957), "Hypertrophy" is increase in size of an organ or tissue, without increase in number of its component units. "Hyperplasia" is increase in size with increase in number of component units . . . "Hypertrophy may be recognized by an increase in unit size. Hyperplasia may be recognized by an increase in unit number and if that increase is still taking place when the tissue is studied, mitoses will give visible evidence of the proliferation."

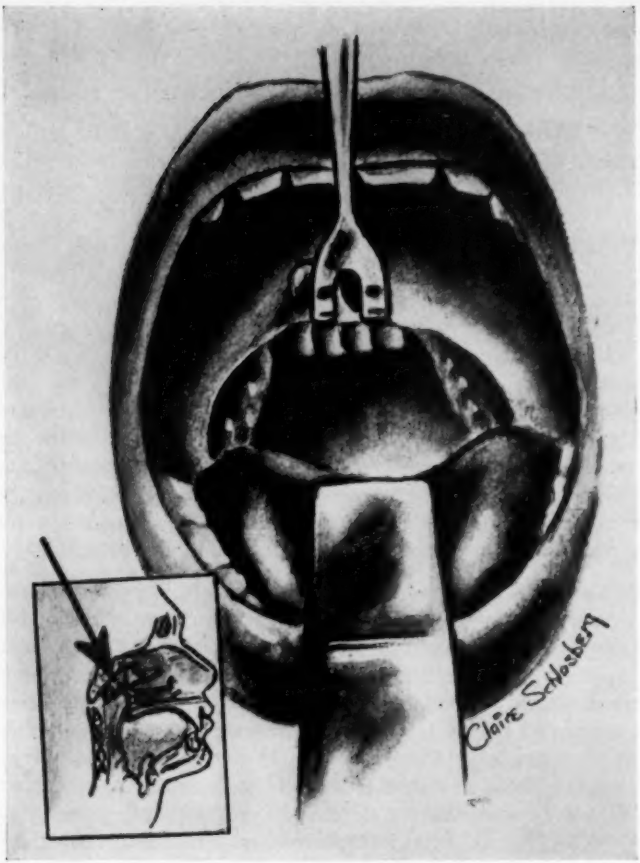

Fig. 3. Adenoiditis in a child. Hypertrophy or hyperplasia of nasopharyngeal lymphoid tissue indicated by arrow in insert.

pletion. This is an erroneous assumption, because in a study of 140 autopsy specimens, Szanto and I[15] found the incidence of nasopharyngeal lymphoid hyperplasia in older persons to be relatively high; in fact, it was not an especially uncommon finding in specimens from subjects after the sixth or seventh decades of life (see Fig. 4). That this observation was not

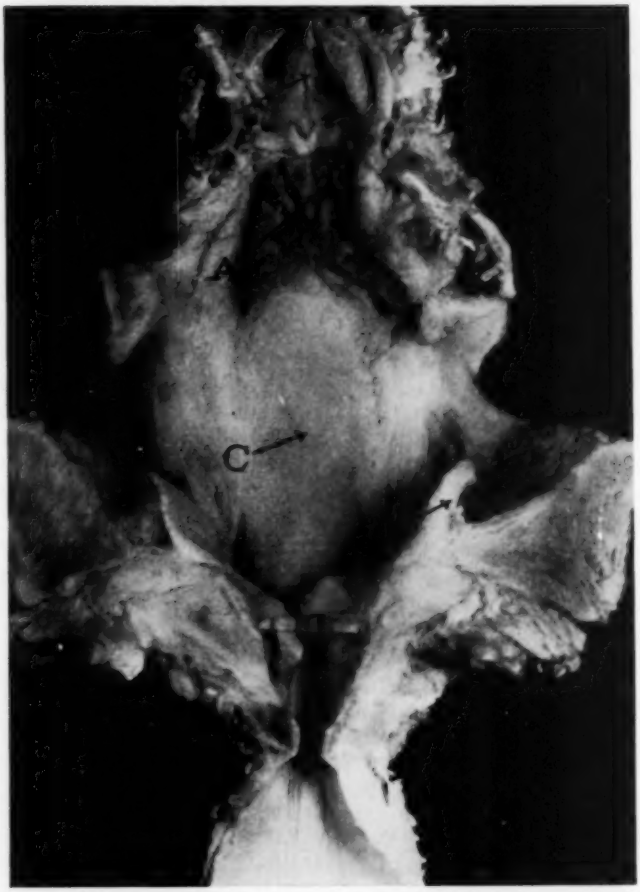

Fig. 4. Specimen from a woman, aged 75 years. A, nasopharyngeal tonsil showing pronounced hyperplasia; B, septum nasi; C, posterior wall of pharynx; D, epiglottis.

new, however, is attested by the reports of earlier workers. Chiari,[16] as early as in 1903, recorded that hypertrophy of the nasopharyngeal tonsil may develop in persons over 60 years of age. Despite the possible clinical significance of this tissue in older persons, it has not attracted the interest it merits.

This may be due to the fact that frequently the process is completely devoid of clinical symptoms; on the other hand, the hyperplastic tissue may be completely overlooked merely because the age factor militates against its presence. It was of considerable interest, therefore, to detect lymphoid tissue blocking the eustachian tube orifices in a high percentage of persons of advanced age studied for causes of hearing loss.[17] The loss, in most instances, erroneously had been ascribed to the age factor.

CLINICAL IMPLICATIONS.

According to Robb-Smith,[18] the disorders of the lymphoid tissue of any region of the body must be divided into two categories: *1.* those initiated by local conditions limited to that region; *2.* those in which there is a generalized abnormality of the lymphoid tissue throughout the body; and, therefore, the lymphoid tissue of the special region undergoes a similar process. "It may be from a clinical point of view," stated Robb-Smith, "that the regional involvement is the presenting symptom of a generalized lymphoid disorder but that in no way justifies its isolation from the point of view of nosography."

"In either category," Robb-Smith continued, "the change in the lymphoid tissue may be reactive; that is to say, the result of stimuli of which we have some knowledge, or idiopathic, in which we have at present no real knowledge of etiology, though we may know much about the morphogenesis of the condition."

Hypertrophied nasopharyngeal lymphoid tissue, regardless of the cause of the pathologic change, often creates or accounts for various clinical problems, more especially in infants and children. In otolaryngology, the main concern is for pathologic processes resulting in the respiratory and auditory organs, difficulties arising largely from the anatomic relationships of the involved structures. Of practical consideration is the question how to deal with these processes when they are encountered. According to Robb-Smith,[18] "if a logical plan is to be pursued in a lymphoid hyperplasia of the nasopharynx, it is necessary to determine whether it is a local

reactive condition in which the treatment is that appropriate to the causative factors, or whether it is part of a generalized lymphoid hyperplasia, even where the nasopharyngeal symptom is the primary manifestation; the treatment is then that of the general disease, and a local eradication is no justification for assuming a cure."

How many of us go beyond elimination of the local process? How many of us consider the likelihood that the nasopharyngeal lymphoid mass is merely the local manifestation of a generalized process? Isn't it reasonable to assume that lack of therapeutic success in some instances can be accounted for by this omission?

THERAPEUTIC CONSIDERATIONS.

Currently available therapeutic methods, surgery and radiation, often fail in producing successful results because their influence is restricted to the local pathologic process. Postoperative recurrence of lymphoid tissue is frequently, but not always, attributable to surgical inadequancy. Failure to arrest the abnormal regenerative activity of the lymphoid organs may be responsible for tissue regrowth. The problem resolves itself in recognizing the absolute necessity of treating the patient and not merely the local disease process.

Logically, the therapeutic approach should emphasize first *prophylaxis*. If it were feasible to control allergy and reinfection of the respiratory tract in infancy and childhood, the incidence of abnormal hyperplasia of nasopharyngeal lymphoid tissue could be rendered negligible. If control of allergy and respiratory tract infection could be made more effective after surgical removal, tissue recurrence might be held to a harmless minimum.

The second therapeutic approach should include an assessment of the patient's general physical state with special reference to possible endocrine dysfunction. Since it is now known that, under certain circumstances, hormonal drugs will reduce or shrink lymphoid tissue mass, the essential concern is for some means to render this effect permanent.

GENERAL COMMENT AND CONCLUSIONS.

Because of its anatomic situation in the upper respiratory tract, the nasopharynx is the main portal of entry for infections and noxious agents. The lymphoid tissue contents of the nasopharynx is part of the general lymphoid system which, it is claimed, plays a major role in the process of immunity.

Lymphoid tissue growth can be stimulated by the administration of certain hormones, by a high calorie-rich diet, and probably by other factors. In animals the growth of lymphoid tissue has been reversed by inanition or by malnutrition, and it may be retarded by exposure to radiation, mustard gas, or by the injection of certain toxic substances. Pathologic changes in lymphoid tissue have resulted from infection, allergy, or their combination, with allergy often playing the cardinal role.[3,14]

Though the age factor militates against its presence, lymphoid tissue is found frequently in the nasopharynx of persons of advanced age, even in the sixth and seventh decades of life.[15]

Disorders of the lymphoid tissue of the nasopharynx may be localized in character, or merely the presenting symptom of a generalized lymphoid disease. In the majority of patients, adequate surgical intervention alone, or surgical intervention followed by radiation has proved effective in strictly localized processes. If these therapeutic measures fail to produce favorable results, the cause often can be traced to inadequate operative procedure, or the neglect of treatment of the underlying systemic disorder, or to both.

SUMMARY.

Present knowledge holds to the belief that the functions of the lymph structures of the nasopharynx are similar to those of the general lymphatic system.

Of the several concepts advanced concerning the growth and involution of lymphoid tissue, the one of a hormonal-lymphoid relationship merits special consideration.

Atrophy of nasopharyngeal lymphoid tissue is not an in-

variable development of advancing age, the incidence of enlargement of this tissue having been found to be relatively high in older persons.

Hypertrophied nasopharyngeal lymphoid tissue creates and causes various clinical problems, the main interest in our specialty centering on complications which develop in the respiratory and auditory organs.

Failures in the management of nasopharyngeal lymphoid tissue often can be accounted for, *1.* by inadequate extirpation; *2.* by lack of regard for a possible generalized lymphatic disorder, and *3.* by absence of an effective method with which abnormal regenerative activity of the lymphoid organs can be controlled or completely retarded.

Theoretically, the ideal would be a method of prevention, adequate control of allergy and reinfections of the upper respiratory tract in infancy and childhood, or following surgical intervention.

Hope rests in advances in hormonal preparations through whose effects the activity of the entire lymphatic system could be adjusted or regulated permanently as the indications warranted.

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GILL MEMORIAL EYE, EAR AND THROAT HOSPITAL.

The Gill Memorial Eye, Ear and Throat Hospital has just completed its Thirty-second Annual Spring Congress in Ophthalmology, Otolaryngology and allied specialties. The attendance was one of the largest in the history of the school with an attendance of 350 physicians and their wives. There were forty-two states, England, Canada and several foreign countries represented. There were twenty-one guest speakers, sixty lectures and closed circuit televised surgery during the five and one-half days of the Spring Congress. In 1960, the Thirty-third Annual Spring Congress will be held from April 1 through April 9.

RAPID EXTERMINATION OF NASAL MYIASIS.*†

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Myiasis is the infestation of the body by the larvae of flies. The screwworm is the larval stage of the *Cochliomyia Americana*, a fly which has a deep greenish-blue metallic color, yellow face, and three dark stripes on the dorsal surface of the thorax. According to Brown,[1] the mature female fly is attracted to some wound or odorous cavity, and only a few seconds are required to oviposit. The eggs hatch within a few hours, and the tiny larvae or screwworms immediately begin to burrow and feed on living tissue. A screwworm is creamy white and is approximately 1.5 cm. in length with 10 to 12 rings or spines encircling its body, giving it the appearance of a screw. Two tiny hook-like projections are mounted on the head, and the breathing apparatus is located in the rear. The maggot, in contrast to the screwworm, is the larval stage of the ordinary blow fly, and it lives on any dead tissue and does not destroy living tissue.

Screwworm infestation in animals is rather common in the South, and is a serious menace to livestock. Fortunately, infestation in the human is rare; however, when it does occur, immediate therapy is mandatory because fatal cases of human myiasis have been reported. Heretofore, various drugs have been used to exterminate the larvae. Chloroform is the agent used most frequently; but, due to the severe irritation and pain from its topical application, an even more effective and less irritating preparation known commercially as Lindane (Hexachlorocyclohexane) is recommended.

Case 1.—A man, aged 62, was seen in the Clinic on July 15, 1950, because of obstruction to the left nasal passage. An examination revealed marked deviation of the nasal septum to the left, so as to completely occlude the airway. The left maxillary sinus was dark to transillumina-

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tion. Submucous resection was recommended, but the patient procrasinated and did not return until October 21, 1950.

Two episodes of postnasal bleeding had occurred during a period of three weeks prior to re-examination. Again, the abnormal nasal septum prevented adequate inspection of the left nasal chamber. A roentgenogram revealed opacity of the left maxillary sinus and loss of definition of the medial wall. On October 26, the left maxillary sinus was explored and a submucous resection was performed. The sinus appeared normal, and erosion through the medial wall of the antrum was not evident. The left inferior turbinate was infiltrated by a rather vascular tumor. Biopsy revealed adenocarcinoma, cylindroma type. The inferior turbinate and tumor tissue was removed and the underlying tissue was electrocoagulated; radium was then applied to the area.

The patient returned on September 29, 1953. Paroxysmal sneezing, nasal irritation, serosanguineous discharge and severe pains in the nose had developed. On inspection, a myriad of screwworms literally filled the nose and were gorging themselves on the nasal mucosa. When the diagnosis was made known to the patient, who was a veterinarian, he became extremely apprehensive. He was aware that the screwworms were destroying living tissue, and that they eventually would erode the cribriform plate, possibly causing meningitis or brain abscess. He informed me that Lindane was the drug of choice for exterminating screwworm infestations in animals, and insisted that he be treated with this drug. Since Lindane is highly toxic, cotton-tipped applicators were saturated with this preparation, and the surplus was removed to prevent drainage into the pharynx. Application was made to the infested nasal mucosa, applying the applicator directly to the larvae where feasible. The screwing motion became retarded, and it was evident that many of the screwworms were dead. By using small nasal forceps, it was possible to remove 62 screwworms. No signs of residual infestation were revealed at subsequent examination.

Case 2.—On September 9, 1957, a transient Mexican laborer complained of severe pain and mild bleeding from the left side of his nose. Three days prior to admission, irritation with slight intermittent bleeding from the nose had been noted. Examination was recorded as follows: "Numerous screwworms are present throughout the left nasal chamber, posteriorly in the floor of the nose. The mucosa appears atrophic and somewhat ulcerated and has a moth-eaten appearance. A little bleeding is evident. Rosenmüller's fossa on the left has a swollen, granular appearance, and several screwworms are noted in the orifice of the eustachian tube." After applying Lindane to the larvae, more than 100 screwworms were grasped with forceps and removed. Those in the eustachian orifice were difficult to eradicate; however, all but one of the screwworms were removed by manipulating a small cotton-tipped applicator under the guidance of a nasopharyngoscope. The following morning, the patient expectorated the lone worm. His convalescence was uneventful.

SUMMARY.

Lindane was found to be a very effective agent for exterminating screwworm larvae from the nasal chambers of our two patients with nasal myiasis.

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PROGRESS IN PAROTID SURGERY.*

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To the surgeon, the criterion of success is the degree to which he achieves maximum removal or disarmament of a pathological process with minimum disturbance of normal function. In dealing with tumors of the parotid gland, which account for the bulk of parotid surgery, anything less than complete removal of the neoplasm results in repeated recurrences, each becoming progressively harder to deal with and correspondingly distressing to the patient. At the same time, a powerful deterrent to competent surgery has been the danger of irremediable damage to the facial nerve.

HISTORICAL BACKGROUND.

The first known attempt to excise the parotid because of a tumor was made by Warren of Boston, in 1804.[28] All early efforts to remove tumors and to protect the facial nerve simultaneously were hampered by insufficient knowledge of the distribution of the nerve, by unrealistic concepts of the gland itself, and by failure to appreciate either the physical structure or the malignant potential of the neoplasms commonly found there. This ignorance led to the practice of a rather gingerly enucleation of such tumors as were brought to the surgeon's attention. This procedure, being only rarely adequate, resulted in such a high rate of recurrence that parotid surgery in general fell into disrepute and came to be avoided as long as possible by both patient and surgeon.

As recently as 1933, McFarland,[40] discouraged by the frequency of recurrences following the enucleation of parotid tumors, voiced the opinion that the patient was as well off without surgery. Nearly a decade later he still found the

*Read at the Sixty-Second Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Hot Springs, Va., March 10, 1959.

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methods of determining prognosis "no more accurate than the flipping of a coin."[41]

Complete parotidectomy, with preservation of the facial nerve, has been practiced for more than half a Century. The first account of this procedure came from Carwadine in 1907,[34] but in a few years other isolated and intermittent reports began to trickle in: Vilray Blair,[32] Barbat,[6] Sistrunk,[59] and Adson.[2] These received very little attention, perhaps because an attitude of pessimism was too firmly embedded; perhaps because the procedures described were not uniformly successful, even when practiced by the men who originated them.

In 1934 a different approach was devised independently by Redon, in France, and by Janes, in Toronto,[32] but it aroused no immediate interest. The following year Bailey performed a similar operation, though apparently he did not report his success or describe his procedure until years later.[5] Its cardinal principles were: first, immediate identification of the facial nerve trunk where it emerges from the stylomastoid foramen, and second, full exposure of the parotid gland. At the same time, he presented what was later termed the first accurate description of the surgical anatomy of the region, though there has since been reason to differ with some of his interpretations.

With the publication of Bailey's work, the pendulum began to swing away from the old policy of avoiding, delaying, and dangerously limiting the surgical attack on parotid tumors. After it was demonstrated that parotidectomy with preservation of the facial nerve was an attainable goal, there developed a trend toward making complete excision of the parotid the routine treatment for all of its tumors. Somewhere between these two extremes probably lies a more satisfactory answer to the long-standing question of successful treatment.

From time to time, various conflicting theories have had their turn at governing the surgical approach to tumors of the parotid gland. Some of these conflicts now seem safely settled, while other theories remain highly controversial. As in most contentions, the chief disagreements probably stem from a lamentable lack of definitive knowledge, especially in

the areas of surgical anatomy of the gland and histology of the tumors found there. Let us consider a few of these concepts and their effects upon methods of treatment.

THEORY OF ENCAPSULATION OF TUMORS.

Belief that tumors of the parotid are generally encapusulated was a large factor in the non-selective practice of enucleation. Its success was doomed by the unusual nature and structure of the great majority of these neoplasms, that group generally termed "mixed tumors." It is now known that the tumor capsule, if one exists, need not be complete;[38,62] that "pseudopods" of the tumor often extend, undetected, into the surrounding tissues,[58] and that even the slightest, probably unnoticed, nicking of the tumor is sufficient to seed the area with cells which may lie dormant for years before finally developing as recurrences.[28] Appreciation of these factors leads to the corollary that infinite care must be taken to avoid spillage of tumor cells, and that one must excise at least a protective margin of normal tissue around the entire periphery of the neoplasm.[46,50]

It has also been discovered that the obvious tumor is likely to be only a fractional part of the whole neoplasm, which Bailey so aptly likened to an iceberg with its greater part submerged.[4] This realization indicated the need for complete exposure of the gland as an assurance that no remnant of the tumor be left behind.

THEORY OF BI-LOBED STRUCTURE OF THE GLAND.

A concept of the parotid as a bi-lobed structure, with the facial nerve lying between the parts in a more or less set pattern, molded the surgical approach for many years, and is still the subject of debate.

Gregoire, in 1912, was first to describe the gland as bi-lobed.[23,30] A few years later McWhorter is said to have demonstrated, to the satisfaction of his audience in 1917, that the facial nerve lies between the superficial and the deep lobes;[64] but again it remained for Bailey to popularize the idea, as he did by referring to the facial nerve as "meat in a parotid

sandwich."[4] Acceptance of this idea probably engendered greater confidence in surgeons who were aware of the need for more than a mere enucleation but yet were fearful of disturbing the facial nerve. Unfortunately, its over-simplification encouraged a less careful dissection than the situation requires, and too often resulted, ultimately, in the very complications which its adherents sought to avoid.

Today, if one reads accounts of the studies made by Ariel,[3] Beahrs,[10,11] Byars,[21] Hurford,[30] Peacock,[37] Slaughter,[60] and Winsten,[67] he must conclude that no matter what the normal anatomy, in the gland distorted by pathology the facial nerve courses through the parenchyma, and for purposes of surgical treatment the parotid must be considered as a single unit, regardless of its embryological structure.

On the other hand, if one studies the reports presented by Brintnall,[18] by Davis and associates,[23] by Levine,[37] and by Utendorfer,[64] he finds equally convincing evidence of the bi-lobed nature of the gland.

It seems possible that the distinct difference in the consistency of the facial nerve, as opposed to the parotid gland, has so facilitated surgical separation of the two organs that on dissection the gland actually simulates a bi-lobed structure; however, it is a dangerous delusion to depend upon lifting one lobe from the other as a means of exposing and protecting the facial nerve during parotid surgery. Only since the investigations reported by Davis and associates in 1956 have we had an adequate concept of the complex anatomy and varied patterns of distribution of this nerve.

Practically, the facial nerve is where you find it. Its safety depends upon diligent seeking, certain identification, and meticulous dissection. Whether the parotid gland consists of one lobe or two would seem to be a question of more academic than practical importance.

CURRENT PROBLEMS.

There are other major points to be debated and important answers to be sought. There is no unanimity as to the extent of surgical intervention that is justifiable in attacking benign

tumors.[29] The value of various diagnostic measures is warmly argued. A great many different classifications of parotid tumors are currently offered. Perhaps, most important of all, and despite the enormous contributions made by Maxwell, Conley, Foote, Frazell, Buxton, Levine, and others, much still remains to be done in the field of histology.

Diagnosis and Evaluation.

On at least one point there is complete agreement: the great majority of parotid tumors manifest themselves as a small, painless lump along the jaw line.

Subsequent growth may be so slow, intermittent, and undisturbing that it remains nearly unnoticed by the patient, who is thereby encouraged to delay in seeking treatment. If the rate of growth is suddenly accelerated, if pain develops, if cervical nodes swell, or if any degree of facial paralysis occurs, then the tumor is almost certainly malignant; but even without such distressing symptoms, the potential of malignancy always exists.

Clinical evaluation formerly was based solely on palpation and inspection, considered in the light of the patient's history, but it may now be supplemented by sialography, though opinions differ as to its value.

The use of sialography as an aid in the diagnosis of neoplasms of the salivary glands was first reported in 1937, by Kimm, Spies, and Wolf.[35] As with every new device, some enthusiasts hailed it as the answer to all problems in the bothersome differential diagnosis of parotid tumors; to such optimists, sialography proved disappointing.[1] Blatt, Maxwell and associates,[15] and more recently Rubin and Holt,[56] have concluded that the procedure can determine whether a tumor is encapsulated or invasive, but not identify the histologic type. Beyer and Blair[13] believe that this is sufficient to differentiate "mixed" tumors from malignant tumors, and to determine whether or not malignant degeneration has taken place; however, some authorities consider the term "mixed" to be synonymous with "benign," while others postulate that a "mixed" tumor may be either benign or malignant. Greater uniformity in terminology would help to settle this point.

After considering the various reports, one may conclude that the greatest possible service rendered by sialography would be a pre-operative warning of the extent of surgery required. This should prevent unnecessarily extensive surgery for both non-neoplastic swellings and for limited, benign growths;[7,55,57] conversely, it should provide a pre-operative admonition of the need for an extensive surgical procedure in the treatment of those tumors which are demonstrably infiltrating.[14,51]

The role of biopsy is debatable. Vellios has asserted that biopsy is always a pre-requisite to effective therapy,[65] but some workers feel that biopsy of no kind is to be tolerated unless accompanied by adequate surgery;[12,50] others maintain that a carefully marked and suitably followed aspiration biopsy is both permissible and helpful.[3,47] Since even the so-called benign tumors of the parotid are said to be the most easily transplanted tissues grown in the human body,[50] and the only ones found, so far, to lend themselves to heterogeneous transplantation, experimentally,[58] one can appreciate the urgent need to prevent spillage and seeding; therefore, if biopsy does precede definitive surgery, there must be considerable care that the biopsy site is adequately extirpated at time of treatment. This applies also to needle biopsy. with the added caution that the site must be effectively marked.

Classification.

Varied classifications of parotid tumors have been proposed.[3,8,9,26,36,38,46,50] For our purpose, perhaps the one suggested by Vellios[65] will be most useful. It is reduced to three groupings:

1. Benign.
2. Malignant, with a long clinical course, and
3. Malignant, with a short clinical course.

While there may be general agreement with the broad form of this classification, opinions differ strongly as to the place of certain growths, particularly the "mixed" tumors, within its framework. Terminology is confused. Varied terms have been used interchangeably, and sometimes it seems that any

neoplasm difficult to identify has been labeled a "mixed" tumor, simply for want of a more accurate name.[8]

Histologic Problem of "Mixed" Tumors.

The puzzling nature of these neoplasms has long occupied the attention of pathologists.[27,42,52] Why do some of these so-called benign tumors seem to undergo a malignant transformation?

Beyer and Blair[13] feel that there is accumulating evidence that repeated surgery and/or irradiation is responsible for such transformation, but Paymaster[49] asserts that there is no proof that surgical trauma ever caused a malignant change and, furthermore, no evidence that a true mixed tumor ever became malignant. Beahrs[12] found that many tumors which were thought to represent malignant change in a mixed tumor were really cylindromas, and pointed out the difficulty of distinguishing between the two; yet he concluded that some mixed tumors do undergo malignant change. Orloff[47] expressed the conviction that the true mixed tumor is uniformly benign, implying that any later manifestations of malignancy could be attributed to a mistaken pathological diagnosis in the beginning. Buxton and associates,[20] during their investigations, found no factor in any benign tumor which seemed conducive to malignant change. In investigating 99 tumors, Foote found no truly benign tumor which metastasized. Levine concluded that "benign tumors . . . do not tend to become malignant;"[37] he inferred that such tumors as appear to do so are really malignant from the beginning, but that their degree of malignancy "is subject to abrupt change." Vellios and Davidson[65] found the question of malignant change in mixed tumors "debatable."

Such a variety of opinion points to the need for further inquiry into the histogenesis of "mixed" tumors in an effort to refine their differentiation. There may be some hitherto unrecognized element in a quiescent mixed tumor which marks it as capable of malignant behavior. Under the present system of identification, it is obviously a grave error to underestimate the potential of any "mixed" tumor. On the other hand, perhaps unnecessarily extensive surgery is now

being practiced because of the general inability to distinguish between the completely benign and the potentially malignant tumors of this type.

Total Parotidectomy vs. Local Excision.

Routine total parotidectomy for all primary tumors has been recommended by Trueblood,[63] Kidd,[34] Redon,[53] Brintnall,[18] Patey,[48] and by Janes.[32] The line of reasoning is thus: first, the record of repeated recurrences and the seeming malignant transformation of supposedly benign tumors makes total extirpation of the gland a justifiable prophylactic measure; second, there are no contraindications, since careful dissection insures as much safety for the facial nerve in this as in a lesser procedure; third, if inadequate primary surgery makes later surgery necessary, the second operation is both more difficult to perform and more likely to result in complications.

At the other extreme, as recently as 1954 Hunter[29] voiced a preference for enucleation followed by irradiation, rather than risk the hazards of parotidectomy.

The conservative view, presented by Devine,[25] Maxwell,[46] Judd,[33] Tabah,[61] Marshall,[43] and Peacock,[50] is that if primary treatment is reasonably early, then a wide local excision generally will prove adequate for all benign growths and for some malignancies. While an occasional report still recommends irradiation as a supplement to conservative surgery,[16] many authorities decry that practice.[3,12,28]

Perzik expressed the opinion of many contemporaries when he asserted that a subtotal parotidectomy is the procedure of choice.[51] Lyle said that a total parotidectomy is indicated only if the lesion is malignant, or if a benign tumor is in the deep lobe.[38] Beahrs considered that the minimal procedure is a superficial parotidectomy, while a total conservative parotidectomy is indicated if the tumor lies beneath the nerve.[9,12]

The difficulty lies in determining beyond doubt, at time of surgery, that the neoplasm is truly benign, that it is incapable of metastasis, and that no microscopic seed of the original growth remains behind.

It is likely that an improved knowledge of the histology of

parotid tumors will make possible a finer distinction among "mixed" tumors, so that in the future fewer total parotidectomies will seem necessary for safety's sake; furthermore, education of the public to the need for seeking early treatment may also help to reduce the necessity for extensive surgery.

Methods of Surgical Approach.

It is generally agreed that the safety of the facial nerve depends upon its exposure and identification without trauma. To accomplish this requires a detailed knowledge of the anatomy of the region, considerable care in operation, and a dependence upon anatomical landmarks.

A variety of such landmarks have been suggested. As early as 1912, Vilray Blair proposed the digastric muscle as a point of reference,[32] but we find no further mention of it in the literature until referred to by Janes in 1943,[21] although it is again cited in contemporary reports.[17,22,44] Seeking the main trunk of the nerve at its exit from the stylomastoid foramen has been a favored custom, though Brintnall and Huffman protest that it may be too difficult to locate, and suggest instead the two bony prominences of the anterioinferior margin of the external auditory meatus and the anterior margin of the mastoid process of the temporal bone as safer guides.[18] Brown[19] prefers to approach the tumor directly and to identify both trunk and branches of the facial nerve as they are incidentally uncovered. Some surgeons have routinely begun with a peripheral branch, which they trace back to the trunk,[2,34,39,59] while others have used the posterior facial vein as a landmark for immediate exposure of the nerve trunk.[21,38] In general, the preferred procedure is first to seek and carefully expose the main trunk of the facial nerve before any other step is taken. Whatever landmarks the surgeon finds dependable will serve his purpose, which is to protect that nerve.

Unfortunately, the VIIth nerve sometimes must be sacrificed when malignancy invades the parotid. Some authorities assert that no attempt should be made to save the nerve when cancer is recognized,[33] while others advocate sacrifice of the nerve

only if the malignancy is either bulky or diffusely infiltrating.[61]

There has been some variation in the type of incision preferred. The "Y", the inverted "W", and the "J" incisions are variously recommended.[9,44,54,66] This would seem to be a matter of personal preference, so long as adequate exposure is achieved with safety.

A RECOMMENDED SURGICAL PROCEDURE.

The surgical procedure which, in my experience, seems most useful, is herewith briefly described.

At present I prefer the "Y" incision (see Fig. 1), since it permits upward retraction of the ear, facilitates access to both the anterior and the retromandibular portions of the gland, promotes adequate exposure of the VIIth nerve, the pterygomandibular space, the mastoid tip, and the sternomastoid muscle, and also aids the completion of a radical neck dissection, if that operation should be found necessary. The anterior component of the incision facilitates the dissection and isolation of the terminal branches of the VIIth nerve, while the posterior portion permits the identification of the parotid gland and reveals its relationship to the sternomastoid muscle. The two components of the incision meet under the lobule at a relatively obtuse angle and then are carried downward along the sternomastoid muscle to the tip of the greater horn of the hyoid bone. At this point the parotid separates easily, unless involved in malignancy, and can be retracted anteriorly.

It is now imperative that the main trunk of the facial nerve be identified (see Fig. 2). I find that the most helpful landmark is the upper border of the posterior belly of the digastric muscle, at the point of its attachment to the mastoid process. The anterior border of the mastoid process itself, above the digastric muscle, is also a dependable guide. By careful dissection to a depth of 1½ to 2 cm. below the skin at this point, the main trunk of the nerve can be located. It will be helpful to remember that the VIIth nerve is the only nerve in this area which runs in a transverse direction and that its width

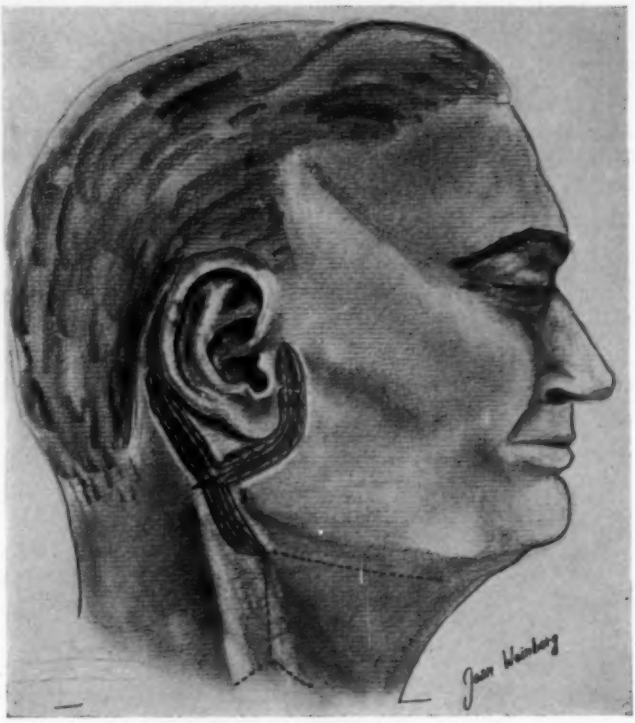

Fig. 1. The Y incision.

at the point of origin from the stylomastoid foramen is from 2 to 3 mm.

As soon as the facial nerve has been exposed, identified, and assured protection, then one must decide upon the extent of resection to be done.

I am convinced that the routine removal of all parotid tissue superficial to the VIIth nerve is the wisest procedure, except when dealing with either very small and superficial neoplasms located near the periphery and no more than 1 cm. in diameter, or else tumors whose greater ramifications im-

mediately become obvious. Such routine procedure offers a protection out of all proportion to whatever additional risk it may carry.

Transient nerve paralysis is to be expected if nerve trauma is incurred at all, but with careful technique the injury will be minimal, and my experience leads me to agree with Martin[45]

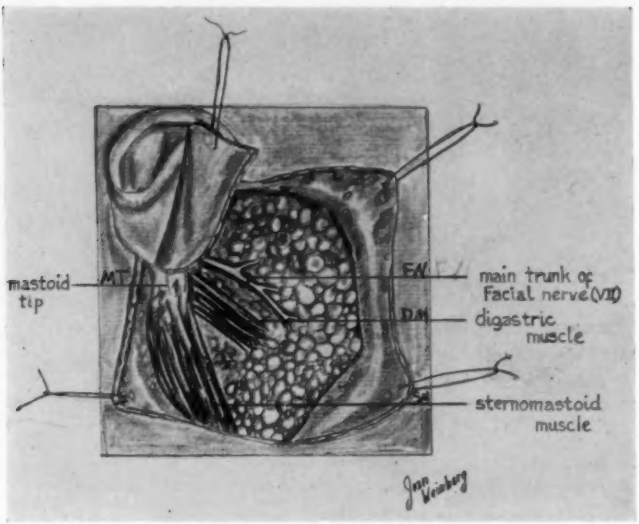

Fig. 2. M.T.—Mastoid Tip; F.N.—Facial Nerve (VII) Main trunk; D.M.—Digastric Muscle; S.M.—Steromastoid Muscle.

that the paralysis will disappear within three or four months; however, one should take care to warn the patient, before surgery, that such a condition may arise and to assure him that restoration of nerve function is confidently expected.

INDICATIONS OF PROGRESS.

Progress in parotid surgery is best illustrated by the striking diminution in both the recurrence rate of supposedly benign tumors and the incidence of postoperative complications.

The recurrence rate has dropped from Wood's conservative report of 50 per cent in 1904[24] to the equally conservative 4.8 per cent announced by Frazell in 1954.[28]

Possible postoperative complications are Frey's syndrome, salivary fistula, and facial paralysis. Incidence of the first two has become negligible,[28] while facial paralysis, once the dreaded concomitant of even the most superficial parotidectomy, is now a rare accident in the initial surgery of any benign growth. It is a distinct mark of progress that today the facial nerve never need be seriously and permanently impaired, except by deliberate intent such as during the pursuit of a deep-seated malignancy.

It would seem fair to conclude that noteworthy progress in parotid surgery began with the practice of bold exposure of the gland itself, safeguarded by immediate isolation of the facial nerve, and motivated by a growing appreciation of the structure and potential growth patterns of the neoplasms found in this area.

With continuing improvement in surgical techniques and increasing knowledge of histology, it is reasonable to expect that the recurrence rate of benign parotid tumors should approach the zero percentile.

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CORRECTION.

The paper entitled "Regression of Hearing Improvement after Mobilization of Stapes" published in the March, 1959, issue of THE LARYNGOSCOPE, should bear the name of Dr. Richard J. Bellucci only. Dr. Dorothy Wolff wishes it to be known that she in no way helped to assess the results of this surgery and, therefore, her name should not appear on this paper.

MASTOID PNEUMATIZATION: A CASE WITH INTERESTING DEVELOPMENTAL AND PHYLOGENETIC ASPECTS.*

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It is generally accepted that the mastoid antrum is the mother of all mastoid cells. It is known, however, that hypotympanic cells invade the mastoid on occasion. It is the aim of this paper to present a case apparently demonstrating a hypotympanic-mastoid tip cell tract, and to discuss this tract regarding incidence, anatomy, clinical aspects, and radiological and phylogenetic considerations.

INCIDENCE AND ANATOMICAL CONSIDERATIONS.

A tympanomastoid tract is not an anomaly or mutation. Its anatomical course and relations are too constant, and as will be discussed later, there are phylogenetic aspects which support this view. Wittmaack[1] in 1918, described a tract of cells in infant temporal bones which he believed was concerned in pneumatization of the mastoid tip from the middle ear. Almour[2] in 1933, confirmed this in adult temporal bones, finding such a tract in 100 per cent of 24 well pneumatized adult specimens. He described the course, from the tip to the hypotympanum, as follows: "From the tip the course of the cell is upward and inward along the posterior canal wall, and then inward overlying the lateral sinus until it reaches an area located posterior to the jugular bulb. It then runs underneath the facial nerve to emerge in the middle ear at the innermost part of the junction between the posterior and inferior tympanic walls."

Bast and Forester[3] in 1939, studied histologically 96 specimens and observed that in only a few specimens mastoid air

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cells opened low into the middle ear directly anterior to the facial canal. Lindsay[4] in 1940, dissected 100 temporal bones in a study directed primarily to the petrous pyramid, and described pneumatization of the infralabyrinthine area in 25 per cent. In 13 per cent there was a tract from the hypotympanum to the mastoid, with the exact portion of the mastoid not given. In 3 per cent the hypotympanic tract led to the infralabyrinthine area only. In 8 per cent there was a tract from the mastoid to the jugular bulb, or round window niche region, but not communicating with the hypotympanum proper. He concludes, in a clinical consideration, that in the majority (with no hypotympanic drainage) there is a greater tendency for suppuration to be walled off, than in those cases where there is an opening to the hypotympanum. Singleton[5] in 1944, dissected 100 temporal bones and found nine tympanomastoid tracts. He stated that usually the retrofacial and infralabyrinthine cells arise from the antrum and extend downward and inward to their destination below the posterior semicircular canal and adjacent to the jugular bulb; but that occasionally these cells arise from the middle ear cavity, pass medial to the facial canal and reach the same location.

Farrior[6] in 1949, stated that the hypotympanic cells posteriorly may extend medial to the facial nerve and are usually responsible for the retrofacial and infralabyrinthine cellular development. He further noted that hypotympanic pneumatization is highly developed in the anthropoid apes, and is rudimentary in man. Attention is called to the following observations from the above. There is variation in the reported incidence of hypotympanic pneumatization of the mastoid, ranging from 100 per cent of well-pneumatized bones to merely an occasional case. It is also noted that since Wittmaack's original observations and Almour's confirmation, the mastoid tip itself is not mentioned, though Wittmaack considered this a separate process concerned primarily in pneumatization of the tip. In short, there is some agreement for an appreciable incidence of pneumatization from the hypotympanum for the infralabyrinthine and retrofacial areas, but with some reluctance to recognize further extension to the mastoid tip.

CLINICAL CONSIDERATIONS.

An observation of Lindsay's has already been mentioned above. Almour points out certain clinical considerations of hypotympanic pneumatization of the tip. It demonstrates that it is possible for infection to spread from the middle ear to the tip without involvement of the antrum. It explains those cases with early mastoid tip tenderness in acute purulent otitis media. Similarly, it explains the X-ray study depicting intact cells about the antrum and the greater part of the mastoid in the presence of exquisite tip tenderness. It also accounts for the operative finding of isolated empyema of the mastoid tip cells with the remainder of the mastoid cells intact. He even suggested that with careful study of X-ray plates, close observation clinically and at the operating table, it may be found that isolated tip empyema can be cured without complete exenteration of all mastoid cells. He further states, and keep this in mind when the author's case is presented, that it is conceivable that only the mastoid tip may be pneumatized with the remainder of the mastoid process sclerotic or diploic.

RADIOLOGICAL CONSIDERATIONS.

Schillinger, an otolaryngologist and instructor in otolaryngologic radiology, published interesting studies on pneumatization of the mastoid in 1938 and 1939.[7,8] He attempted a correlation of Roentgen findings to anatomic and clinical concepts. He feels that the classification of mastoids as pneumatic, diploic or sclerotic is neither informative anatomically nor of great assistance otologically. He proposes an anatomic classification based on a roentgenologic study in the first six years of life, during which time the adult pattern can be seen to develop. The fundamental anatomic structure which is well seen on the X-ray film at age two is the peri-antral triangle. This roughly represents the base of the petrous pyramid and contains the antrum, peri-antral cells and labyrinth. It is bounded posteriorly by the groove of the sigmoid sinus and above by the tegmen of the antrum and middle ear. The study is based on the pattern of development of cells as they extend beyond the boundaries of this

basic triangle from ages two to six. Cells are described as originating from the antrum and hypotympanum in three distinct tracts termed squamal, antral and tympano-tip. It is assumed that the squamal cells originate from the squamosal or lateral segment of the antrum and invade the squamozygomatic region. Schillinger's discussion of a squamal tract is remindful of DeLisa's paper on squamositis (1949).[9]

The antral cells make up the largest tract and presumably originate from the medial or petrosal portion of the antrum. The cells of hypotympanic origin, the tympano-tip tract, are considered by Schillinger as a normal development which pneumatizes the tip and anterior face of the mastoid process in a characteristic manner. Each tract is best demonstrated when it develops alone or the tracts are only partially developed, so that there is not too much obscuration by overlapping. When fully developed the tympano-tip tract extends to the emissary vein region posteriorly and is responsible for the broad, pneumatized tip; when it fails to develop the tip is narrow or pointed and is either diploic or partially invaded by antral cells. Illustrative diagrams and roentgenograms are presented in the papers which are quite illuminating. Schillinger classifies mastoids into two main groups: those with normal development and those with defective development. Under defective development are listed the undeveloped mastoid (exhibits no extension beyond the peri-antral triangle) and the partially developed mastoid which presents each of the three tracts, alone or in various combinations.

PHYLOGENY.

It is a general axiom, not always true, that a phylogenetically older structure will appear earlier in the embryo. The ossicles of the middle ear, *e.g.*, are apparent as early as the fourth week (stapes).[10] The mastoid process, on the other hand, is not formed until after birth, being only a small tubercle behind the upper part of the annulus tympanicus (Politzer) at birth. It is not apparent externally in animals below the anthropoid apes, and only attains a true mastoid or breast-like configuration in man. It has been proposed that the mastoid process develops in response to the pull of the

sternomastoid (Alexander)[10] and, or the splenius capitis[11] muscles engaged in maintaining the head in an erect position.

A fascinating phylogenetic development beyond the scope of this paper, but meriting at least summarization, is the transition from the monossicular (columella) system of the premammalian reptiles to the triple ossicular system of mammals. The ossicles are derived from the masticatory apparatus, which on the surface might appear an odd source. From a functional standpoint, however,[13] it might be conjectured that there occurred a mutually beneficial evolutionary change with the masticatory and auditory apparatus not entirely unrelated. The massive jaws of the aquatic premammalian reptiles became secondarily concerned with transmission of sound by bone conduction through an intermediary water and later earth medium. In the transition from a water to an air environment there is temptation to conjecture that there was a concomitant change to, *1.* a middle ear modified for airborne sound transmission via a triple ossicular system, and *2.* simultaneous conversion of the massive jaw mechanism to a lighter one better adapted to function in a less bouyant air environment.

The triple ossicular system is characteristic of all mammals from the lowly duck-billed platypus on up the scale. The epitympanic recess, also a characteristic mammalian structure, is developed of necessity to house the incudo-malleolar mass.[12] In the next higher level, the marsupials, the second purely mammalian characteristic appears, the auditory bulla.[10,12] This is an expansion of the tympanic bone, housing a hypotympanic space hanging from the base of the skull in most instances. In its simpler form it is monocellular, and perhaps serves as a resonating chamber to magnify simple warning sounds from other members of the same species. Both the bulla and epitympanic recess vary from simple one-celled chambers to multicellular systems. In the kangaroo, *e.g.*, the epitympanic recess expands into the squamosa; in moles, the bulla invades the basisphenoid in a multicellular system; in some ungulates, *e.g.* the cow, the bulla is laterally placed and attaches to the petrosa with a multicellular bullar tip and so,

as stated by Eggston and Wolff,[10] corresponds to the human mastoid.

Both Tumarkin[12] (from an unidentified source) and Robb and Palmer,[14] quoting Valsalva (17 Century) as the source, make the observation that when the bulla is small there tends to be compensatory enlargement of the epitympanic recess and its associated air spaces. This suggests a competition, in an evolutionary sense, between the two systems. The bulla is more prominent in lower mammalian forms with the competition reaching a stalemate in the higher ape forms (gorilla), where both systems are maximally developed. Tumarkin's paper shows the variations diagrammatically for various mammals through the primates, with the mastoid of the higher apes equally pneumatized from both sources. This is a transitional stage to man, *i.e.*, the bulla proper is smaller while the accessory multicellular systems become more prominent as the mastoid itself becomes more prominent.[12] In man there is an apparent ascendancy of the epitympanic recess accessory cell system concomitant with the further development of the mastoid process.

In the highest order of mammals, the primates, the lemurs (a lower form) have a large monocellular bulla with a narrow neck, suggesting a Helmholtz resonator. In the New World monkeys[12,15] there is a flat mastoid, a tympanic ring without a bony canal and a bulla at its union with the petrosa. There is a well developed accessory air cell system both in the petrosa and the mastoid region. In the Old World monkeys there is a transitional stage to the structure found in man. The bulla proper diminishes in size, and a mastoid bulge is present. A bony external canal is formed, and the cell system is still better developed. In the gorilla the mastoid is a flange-like plate with the digastric groove exposed to view in the lateral aspect.[14] The gorilla particularly has a remarkably widespread cell system ramifying throughout the mastoid, squamosa and petrosa with the labyrinth and carotid, *e.g.*, suspended in air cells. Hoffman[15] points out that in the anthropoid apes extensive, symmetrical pneumatization is consistently present. In man the accessory air space system rarely attains the extent found in the apes, and is usually greatly reduced,

especially as regards pneumatization in the petrous portion; also there is often asymmetry between the two sides in man. In addition, diploic and compact temporal bones are more common in man. Whether these are secondary to infantile or prenatal inflammation (Wittmaack) or normal for that individual (Cheatle, Diamant, etc.), is not pertinent to the scope of this paper.

From paleontology it is learned that the anthroproid apes date back 65 million years.[15] Peking man, hailed on discovery in 1936 as the "missing link," dates back only 600,000 years.[16] His teeth and mastoid configuration are definitely anthropoid; however, his larger brain case and his ability to make fire and use tools are humanoid. Neanderthal man dates back 200,000 years and his mastoid is described as intermediate between the apes and man.

From the above it can be said that a tympano-mastoid tract is not accidental and has precedent in phylogeny. It may be atavistic in the sense that it reverts to the time when the epitympanic and hypotympanic cell systems were more evenly matched in their struggle for ascendancy.

CASE REPORT.

Present Illness: J.D.C., 26-year-old white male IBM machine operator, was admitted Aug. 7, 1956, with a history of intermittent drainage from the right ear since June 1, 1956, and with a right facial paralysis of one week's duration. The ear had been treated by unidentified ear drops but with a persistent discharge abating only one week before admission.

Past and Family History: There is a history of right-sided mastoiditis at age 6 but no mastoid surgery. Three of four siblings had ear infections in childhood.

Physical Examination: A right facial paralysis was present with slight motion persisting in the eyebrows and angle of the mouth and with good muscle tone. The left drum was markedly retracted. The right drum showed a friable, irregular polyp occupying a posterior *pars tensa* perforation. There was no mastoid tenderness or swelling. Hearing in both ears was roughly equal with ordinary conversation readily heard. Audiogram showed excellent bone conduction bilaterally, flat curves, with a 30 db loss for the conversational range on the left and a 25 db loss on the right. Fistula test was negative.

Laboratory: Essentially normal blood count, urinalysis and serology. Mastoid X-rays were reported as follows: "Poor pneumatization on the left and incomplete pneumatization on the right." More detailed retrospective review by the author reveals the left mastoid largely diploic. On the right side there were small cells in the peri-antral region with a limited distribution beyond the triangle. There were large, intact, thin walled cells in the tip region (see Fig. 1).

Course in the Hospital: The polyp was biopsied and reported as a nonspecific granuloma. The facial paralysis cleared under antibiotic therapy, but the polyp remained unchanged. Two weeks after admission a mastoidectomy was performed. The external canal was narrowed. A small space containing pus was encountered in drilling toward the antrum. The mastoid was infantile in type with a high, anterior and deep antrum, which was found with some difficulty. The antrum was a small, flat slit, with a sharp sloping roof and was 3 mm. in height. It contained a drop of pus. The lining was slightly thickened but without trabeculation or visible opening other than the aditus. The short process of the incus was slightly granular in appearance. The lateral sinus was en-

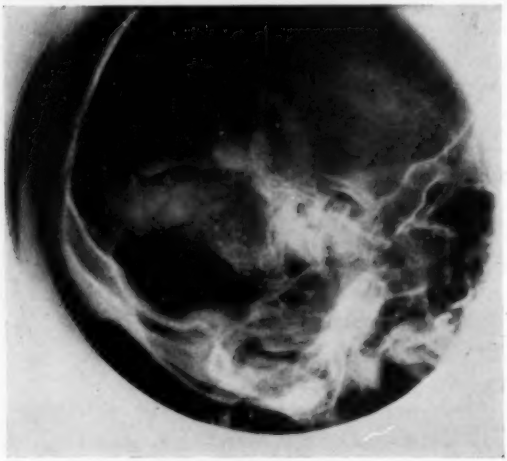

Fig. 1.

countered 12 mm. from the posterior canal wall. Toward the tip large cells with a normal lining were encountered. The aditus was uncapped and the epitympanum seen to be free of disease. The posterior canal wall was partially taken down to allow drainage for the diseased portion of the mastoid. The tip cells were left largely undisturbed, as was the middle ear proper. No graft was applied. The cavity was lightly packed open.

Postoperative Course: A sterile mastoid dressing was maintained for six weeks. By the end of four weeks the exposed and opened tip cells were seen to have sealed themselves off gradually by primary healing. A smooth glistening epithelium had extended from the external canal to almost cover. The polyp disappeared and a clean dry perforation remained through which a clean incudo-stapedial joint could be visualized. When last seen 25 months after operation, there was a flat 30 db curve on the right and with a dry, clean perforation. A cigarette paper prosthesis gave no improvement in hearing.

This was an infantile mastoid with partial, small cell pneu-

matization of the greater portion of the mastoid draining via a hypoplastic antrum but with a well developed tip cell system. It approaches Almour's hypothetical case of a normal tip cell system draining into the hypotympanum and a diploic or sclerotic mastoid otherwise. In the above case it must be conceded that hypotympanic origin is based on indirect though considerable evidence. Had the author been familiar with

Fig. 2.

Schillinger's work at the time this case presented itself, could he have justifiably predicted the pathological anatomy on the basis of the X-ray? It may be of interest that the patient's mother has one mastoid with only the tip cell system developed but with normal pneumatization on the other side (see Fig. 2).

SUMMARY.

There is anatomic, clinical, radiological and phylogenetic evidence for a double origin of mastoid cells. Retrofacial, infralabyrinthine and mastoid tip cells may be pneumatized

from the hypotympanum. The incidence of hypotympanic origin of mastoid cells, especially the tip cells, is not well established but is probably greater than is generally appreciated. Attention is called to Schillinger's work with X-ray studies which warrants further correlation with anatomical studies.

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Veterans Administration Center.

FIBROUS DYSPLASIA OF THE TEMPORAL BONE.*

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New Orleans, La.

This report is concerned only with monostotic fibrous dysplasia of bone. About this, Schlumberger[3] says: "Histologically the primary component of the bone lesion is connective tissue, fairly well vascularized and frequently arranged in interlacing bundles and whorls. Within the connective tissue, and most abundant at its periphery, are trabeculae of partly calcified newly formed bone."

In Schlumberger's[3] report of 67 cases of fibrous dysplasia involving single bones, there was only one case of involvement of the temporal bone. Brunner[1] reports a case of disease of the malar bone with apparent exostosis of the external auditory meatus; biopsy was done of the malar bone, and the mass in the canal of the ear and fibrous dysplasia was demonstrated in each instance. Towson[4] reports a case of fibrous dysplasia of the mastoid in a colored boy 14 years of age. I have found only these three biopsy proven cases of fibrous dysplasia of the temporal bone in my search of the literature; however, it may be less rare than the literature indicates, since fibrous dysplasia of the temporal bone may have been reported under other designations. Cooke and Powers[2] point out that as many as 33 different titles had been used to describe this entity prior to the introduction of the term "fibrous dysplasia" by Lichtenstein in 1938.

Fibrous dysplasia of the mastoid process is an expanding bone lesion, and among its first clinical symptoms may be an occlusion of the external auditory meatus by what appears to be an exostosis. This was true in two of the three cases I have found in the literature, and it was the initial symptom in the case reported here by me.

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*Presented at the meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society, Atlanta, Ga., Jan. 24, 1959.

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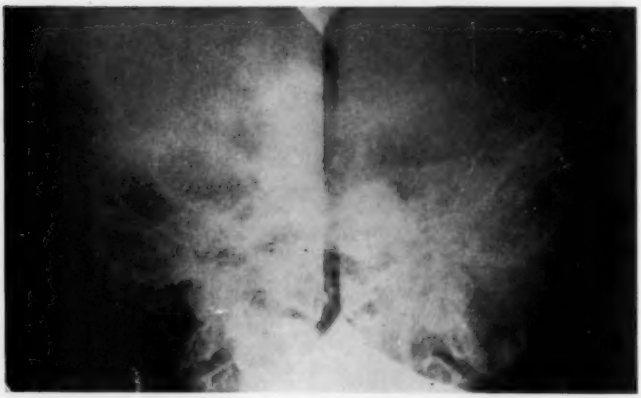

Fig. 1. Preoperative X-ray study showing gross enlargement of right mastoid and petrous.

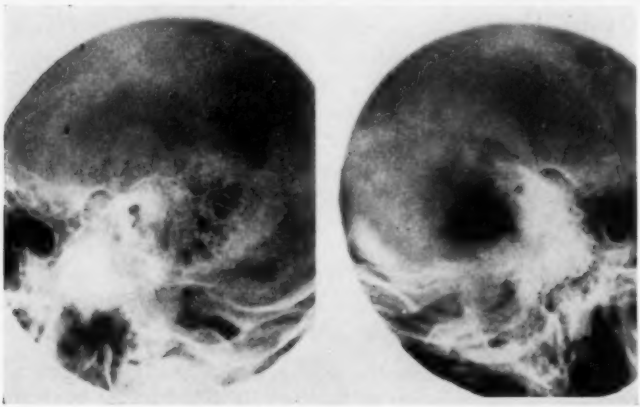

Fig. 2. Postoperative lateral view of mastoids showing surgical defect of right mastoid.

A.S., Jr., was first seen August 9, 1950, at age 21 with the complaint of impaired hearing in his right ear, which was discovered when he was rejected for the Navy in 1948. The right external auditory meatus was obstructed by what appeared to be an exostosis. There was no history of trauma to the ear or the head. His physical appearance was that of a healthy 21-year-old man. The Weber test lateralized to the right ear.

At X-ray examination the petro-mastoid portion of the right temporal bone showed its original contour, but was grossly enlarged and contained

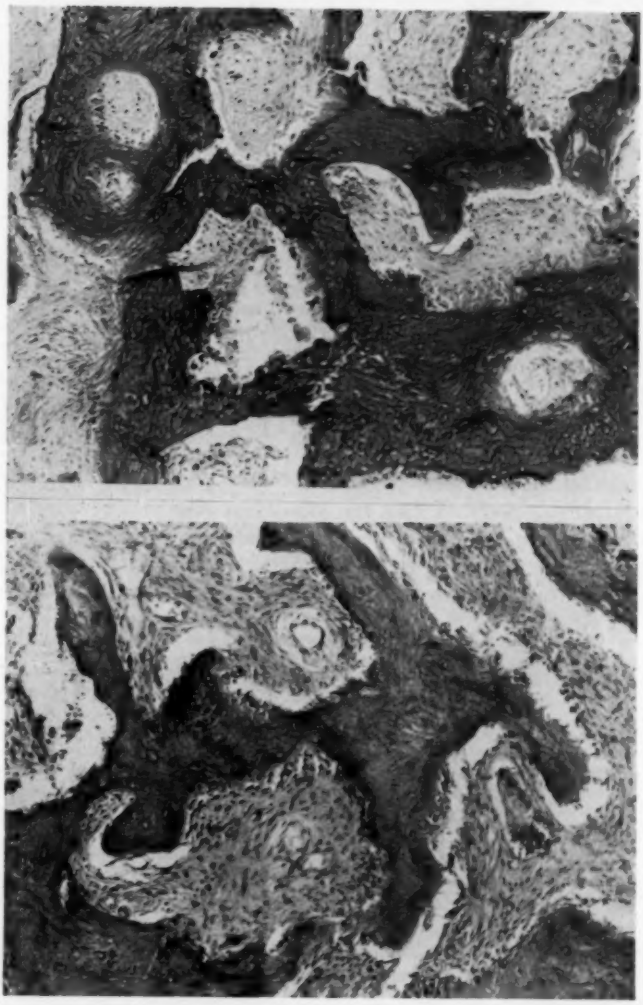

Fig. 3. Photomicrographs showing fibrous dysplasia.

no pneumatic spaces. Skeletal survey showed no evidence of any other bone lesions. Serum calcium and phosphorus were within normal limits. In August, 1950, a bony mass was removed from the right ear; however, the ear drum could not be seen. A lesion recurred and was removed in March, 1951, and a year later the ear was again completely obstructed by recurrent lesions. The pathologist reported the bony tissue removed at these operations as compatible with osteoma.

On June 27, 1956, the patient came into my office supported by two men and reported that he had had vertigo for four days on walking or sitting up but none on lying down. He obviously had some labyrinthine irritation, but there were no new objective findings and X-ray examination was essentially the same as before.

July 3, 1956, right mastoidectomy was done by the postauricular approach. The mastoid process was a smooth and massive structure. The cortex was removed with a gouge and appeared to be bone of normal density for this structure. The underlying bone structure had a peculiar granular texture, and showed no pneumatic spaces; it bled rather freely. It was scooped out easily with a large curette, and three large cavities were found containing cholesteatoma and some fluid but no landmarks. There was no escape of cerebro-spinal fluid. The matrix of the cholesteatoma was not removed. One cavity of an estimated capacity of about 25 cc. was created from these three cavities, and the deepest part of the cavity was estimated as two inches from the mastoid cortex, probably as deep as the internal auditory meatus in the posterior fossa. The walls of the deepest cavity were bony, except that deeply and posteriorly the wall was densely membranous and non-pulsating, but five months later and for a period of about a month, pulsation was observed in this area. The cavity was packed and the posterior wound sutured.

Microscopic examination of tissues by Dr. A. J. Hertzog, Chief of Pathology at Touro Infirmary was reported: *1.* Cholesteatoma; *2.* Fibrous dysplasia of the mastoid bone. The slides were reviewed by Dr. C. E. Dunlap, Chief of the Deptartment of Pathology at Tulane University, and Dr. Dunlap concurred in the diagnosis.

The patient's postoperative course was good and he rapidly lost his vertigo. After the wound cleared up, the right ear drum, which had been covered by the matrix of the cholesteatoma, was found intact and his hearing in the right ear showed a loss of about 30 db for the conversational frequencies. At the present time examination of the right ear shows a large cavity which is dry and completely epithelialized; the ear drum is present and intact. Vertigo and horizontal nystagmus to the right can be produced by lightly touching a spot in the depth of the cavity with a cotton applicator. I suspect that this is from contact of the probe with the saccus endolymphaticus, but it may be that he has an undiscovered fistula of the posterior semicircular canal.

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Section on Laryngology, Otology and Rhinology.

Atlantic City, June 8-12, 1959.

(Rather than assigned discussers, each paper, except those of the Guest of Honor and the Chairman, will be followed by an open discussion period; questions and comments from the floor are encouraged.)

Tuesday, June 9—2 P.M.

PROF. DR. H. H. NAUMANN, Würzburg, Germany—
Intravital Observations of the Nasal Mucous Memberane (Film).

GEORGE M. COATES, M.D., Philadelphia, Pa.—
Address of Guest of Honor.

GORDON D. HOOPLE, M.D., Syracuse, N. Y.—
Modern Otolaryngology.

F. W. DAVISON, M.D., Danville, Pa.—
Laryngeal Obstruction in Children.

BEN T. WITHERS, M.D., Houston, Tex.—
Facility in T&A Management: Conclusions from 2400 Consecutive Cases.

JOSEPH H. OGURA, M.D., St. Louis, Mo.—
Cancer of the Pharynx, Larynx and Upper Esophagus; Surgical Aspects.

BUSINESS MEETING:

Report of Section Delegate to AMA House of Delegates—
Gordon F. Harkness, M.D., Davenport, Ia.
Dean M. Lierle, M.D., Iowa City, Ia., Alternate.

Report of Section Representatives to Board of Governors of American College of Surgeons—
Roderick Macdonald, M.D., Rock Hill, S. C.
Edley H. Jones, M.D., Vicksburg, Miss.
John J. Conley, M.D., New York, N. Y.

Report of Nominating Committee—
F. W. Davison, M.D., Danville, Pa.
Louis E. Silcox, M.D., Philadelphia, Pa.
John D. Singleton, M.D., Dallas, Tex.

Wednesday, June 10—2 P.M.

J. W. HAMPSEY, M.D., Pittsburgh, Pa.—
Current Concepts of Etiology and Management in Otolaryngic Allergy.

VICTOR R. ALFARO, M.D., Washington, D. C.
Address of Section Chairman.

HENRY S. KAPLAN, M.D., San Francisco, Calif.—
New Horizons in the Radiotherapy of Malignant Disease.

JOHN A. KIRCHNER, New Haven, Conn.—
Facial Bone Injuries.

M. R. HIMALSTEIN, M.D., Bay Pines, Fla.—
Obliterative Frontal Sinus Surgery Using Gelfoam.

J. M. RAVID, M.D., New York, N. Y.—
Malignant Melanoma of the Nose and Paranasal Sinuses, and Juvenile Melanoma of the Nose.

Wednesday Evening, June 10—6:30 P.M.

Social Hour and Dinner Honoring the Guest of Honor of the Section, Dr. George M. Coates.

Thursday, June 11—2 P.M.

JOSEPH L. GOLDMAN, M.D., and HARRY ROSENWASSER, M.D., New York, N. Y.—
Current Concepts of the Management of Otitic Infections.

MERLE LAWRENCE, Ph.D., Ann Arbor, Mich.—
Ear Physiology.

EDWARD W. HARRIS, M.D., Columbus, O.—
Symptoms Referable to the Eustachian Tube.

GEORGE KELEMEN, M.D., Boston, Mass.—
Maternal Diabetes and Congenital Deafness.

JOSEPH SATALOFF, M.D., Philadelphia, Pa.—
Stapes Mobilization with Long-Standing Otosclerosis.

HOWARD P. HOUSE, M.D., Los Angeles, Calif.—
Polyethylene in Middle Ear Surgery.

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(Secretaries of the various societies are requested to keep this information up to date).

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Meeting: Atlantic City, June 8-12, 1959.

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Meeting:

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Annual Meeting: October 10, 1959, Belmont Hotel, Chicago, Ill.

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Meeting: Sheraton-Brock Hotel, Niagara Falls, Ontario, October 9-10, 1959.

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Meeting:

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Chairman of Otolaryngology Section: Dr. Robert W. Godwin.
Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

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Meeting:

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Meeting:

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Meeting: Palmer House, Chicago, Ill., October 11, 1959.

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Meeting:

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President: Dr. Paul Holinger, 700 No. Michigan Blvd., Chicago, Ill.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Seventh Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Miami, Fla., March, 1960.

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